ANNALS
OF
OTOLOGY, RHINOLOGY
AND
LARYNGOLOGY

VOL. 43. DECEMBER, 1934. No. 4.

LXXVIII

THE HISTOLOGY OF THE EPITHELIUM OF THE PARANASAL SINUSES UNDER VARIOUS CONDITIONS.*

JOHN STEPHENS LATTA, PH. D.,

AND

ROY FRANKLIN SCHALL, M. D.,

OMAHA.

In spite of the fact that literature dealing with the gross and microscopic characteristics of the accessory nasal sinuses has been rapidly accumulating during the past few years, it is difficult to find any adequate descriptions of the histologic features of these organs. The great bulk of related investigations have described these sinuses as observed under markedly pathologic conditions or in some cases as seen under experimental conditions, but without having unquesably established the histologic appearance of the sinus membranes under average conditions. Practically all of the literature above referred to has been the product of rhinologists, relatively little attention having been given the subject by those working in the field of anatomy and histology. The most exhaustive investigation of the anatomy of these structures has been that of Schaeffer (1919), but in none of his reports has he included detailed description of the histologic characteristics of the mucous membrane lining them. It was with the hope of being able to present a more adequate description of these tissues that the present study was undertaken.

*From the Department of Anatomy, College of Medicine, University of Nebraska, Omaha, Neb.

The obvious fact that the epithelium and, secondarily, the underlying connective tissue of the mucous membrane of the nose and its appended sinuses is subject to tremendous environmental changes from time to time, which we believe to be particularly true of sinuses because of their peculiar anatomic relationships to the nose, makes it exceedingly difficult to establish conditions which could be called normal, and, therefore, to determine histologic features which could, with any assurance, be stated as those of a normal sinus membrane. Such attempts to establish limits of normal variation, we believe, would be more successful in some other mammalian forms in which there are apparently more efficient means for dealing with environmental changes without marked change in tissue character and, consequently, in which nasal and sinus disease are less frequent than in man.

On the other hand, the fact that environmental changes in these locations may be so great and the corresponding tissue changes so pronounced offers in the sinus membranes a rather unusual opportunity for the investigation of the expression of the potentialities resident in the various tissues concerned. In this paper we shall largely confine ourselves to a study of the potentialities of the epithelium in the paranasal sinuses for hypertrophy, hyperplasia, and regeneration, etc., as seen in tissue secured under a great variety of conditions.

MATERIALS AND METHODS.

We were very fortunate in being able to secure an abundance of fresh human material removed during radical sinus operations. Incidentally, the indications for operative intervention were quite variable and the histologic conditions encountered were correspondingly variable. Aided by the use of a large hollow auger drill,* we were also able to secure several sinus specimens at postmortem in which there had been no indication of upper respiratory or paranasal sinus infection. For comparison with the material so obtained, we also made preparations from sinus mucous membranes from the calf, dog, cat and rabbit, including maxillary, frontal and ethmoid sinuses in the calf; maxillary and frontal sinuses in the dog and cat; and maxillary sinuses in the rabbit.

All surgical specimens were immediately placed in the fixing agent, either Maximow's modification of Helly's fluid or Bouin's picro-aceto-formol, the former being used whenever possible. The autopsy specimens were fixed as soon postmortem as the necessary permission could be secured. The other mammalian material was,

*This drill was designed by Dr. F. L. Dunn and D. Rhea and described by them in Arch. of Path., Vol 15, p. 255.

of course, fixed immediately postmortem. After fixation and washing (Maximow's fluid) dehydration was accomplished with alcohol and clearing, either in xylene or cedar oil. The material was then infiltrated in, first, 40 to 42 degrees paraffin, and then either in 56 to 58 degrees or 60 to 62 degrees paraffin in which it was later embedded.

Whenever possible, sections were cut at five microns. In two or three cases where the material was difficult to handle, sections were secured at either eight or ten microns.

Delafield's hematoxylin followed either by azure II-eosin or by Mallory's aniline blue connective tissue stain were used routinely. Supplementary stains used were Weigert's or Heidenhain's iron hematoxylin, Weigert's copper hematoxylin, Wright's followed by Giemsa stain, Dominici's eosin-orange G-toluidin blue mixture and Jenner's stain. In a few instances muchematein or mucicarmine were also employed.

In using the first two stains we made slight modifications. Delafield's hematoxylin was used full strength for from two to three minutes, the slides then being removed to distilled water, which had been slightly acidulated with either hydrochloric or acetic acid, for thirty minutes. When azur II-eosin followed, the sections were stained for thirty minutes in a mixture of 10 cc. of 1 per cent azur II and 25 cc. of 1 per cent water soluble eosin. When Mallory's connective tissue stain followed, the sections were stained in the acid fuchsin (2 per cent) for four or five minutes and then in the aniline blue-orange G-phosphomolybdic acid mixture for a period of only three or four minutes. In both cases differentiation was made in 95 per cent alcohol, xylene used for clearing, and neutral xylene damar for mounting.

DESCRIPTION OF MATERIALS.

In general, the mucous membrane of the paranasal sinuses has been described as being composed of a loose connective tissue covered by a ciliated columnar epithelium (Turner, 1901; Skillern, 1913). Schaeffer (1928), in a somewhat more detailed description, gives its composition as a pseudostratified ciliated epithelium lying upon a tunica propria which blends smoothly into the periosteum of the surrounding bones.

In the present study we have chosen to discuss the histologic features of each of the paranasal sinuses separately. In describing variations which we have found we did not attempt to differentiate

sharply between normal physiologic and pathologic conditions, which would be extremely difficult if not impossible.

THE MAXILLARY SINUS.

The mucous membranes of this sinus, which showed the least epithelial differentiation and in which the underlying connective tissue by its relative thinness and cellular simplicity gave no evidences of pathologic processes, were assumed to be within the range of normal structure. In these cases the covering epithelium was composed principally of relatively tall columnar ciliated cells, between the bases of which lay numerous irregularly shaped basal cells not extending to the free surface. Only occasionally were mucus secreting goblet cells to be found between the columnar ciliated cells.

The tall ciliated cells varied somewhat in size but always extended from the free surface to the basement membrane, the basal ends being usually very tapering because of the large number of interposed basal cells. The ciliated ends completely covered the lumen surface except for the small openings of the scattered goblet cells. The cilia of these cells were quite long, ranging from six to ten microns in length, attached to densely stained basal granules, which were very near the peripheral limiting membrane (Figs. 4 and 5). The cytoplasm of these cells appeared quite granular, the granules being finer in the basal portion and coarser nearer the surface. In material stained with Heidenhain's iron hematoxylin a quite definite cytoplasmic network was seen. The oval nuclei of these cells stained rather densely, the chromatin massing in clumps. A nucleolus could usually be found.

The basal cells were easily distinguishable from the ciliated cells in that their cytoplasm was much less granular and more homogeneous. The nuclei in these cells were very similar to those of the ciliated cells but were usually spherical and more vesicular. These cells have been considered (E. A. Schafer, 1912) as progenitors only of goblet cells, in that he found them to contain mucinogen in all stages of their growth. This finding could not be verified in our studies, for no mucinogen granules were demonstrable in basal cells after staining with mucicarmine, muchematin or Wright-Giemsa stains.

The goblet cells which were present in variable numbers presented great variety in size and form, depending upon their stage of development. When fully formed these cells were tremendously distended peripherally with contained secretion, the basal process remaining very narrow, and contained a very small compact nucleus easily distinguished from those of the ciliated or basal cells. These

nuclei were often so pycnotic that nuclear details were completely obscured. In goblet cells in which much of the mucin had been washed away during preparation a granular network remained, which stained intensely with Wright-Giemsa or mucicarmine stains.

The epithelium composed of the cell types just described was supported by a loose connective tissue with a large proportion of collagenous fibers with numerous scattered fibroblasts. Elastic tissue stains revealed practically no elastic fibers except as occurring in the walls of blood vessels (as noted by Schaeffer, 1928), which were quite numerous, especially close to the epithelium. Weakly secreting mucus glands were found in this connective tissue. They were neither at all numerous nor complicated in structure.

In the maxillary sinuses of the other mammalian forms studied the epithelium was also found to be of a pseudostratified ciliated columnar variety. In the dog, cat and rabbit these cells were quite tall, whereas in the calf they were almost cuboidal in shape (Figs. 2, 8 and 14). In the case of the dog and cat no goblets were found, the surface being completely covered with ciliated cells, while in the calf and rabbit only a few were found.

The appearance of the connective tissue in these comparative forms corresponded closely with that found in man. Glands, however, were evidently more numerous, producing a weak mucus secretion. Subepithelial lymphatic nodules were frequently encountered in these cases also.

Returning to the human material, many variations were found in the nature of the covering epithelium, even though the underlying connective tissue was unchanged. In these and in the various other preparations studied the potentialities resident in the epithelium were well shown. In a few places in different specimens there were found small areas from which the epithelium had been completely lost. At the edge of such areas the adjacent columnar ciliated cells showed a tendency to spread out and close over the denuded surface. Along the exposed sides of such cells cilia were usually present, although not entirely to the basement membrane.

In some specimens areas were to be found in which the superficial epithelial cells had been sloughed, leaving only the layer of basal cells. The lack of differential change found in these cells suggested that this sloughing might have occurred during manipulation of the tissue rather than any degenerative process.

In some cases, especially where large numbers of fully developed goblet cells were present, the evacuated goblet cells were apparently

in the process of sloughing away, leaving the basal cells only adherent to the underlying connective tissue.

In another portion of one specimen in another part of which typical epithelium was present, the covering was composed only of a single layer of (basal) cuboidal cells, some of which bore a few poorly developed cilia (Fig. 2). Most of the cells which bore no cilia possessed cytoplasmic protuberances, in some of which extensions numerous very deeply stained (iron hematoxylin) granules were present. The granules were quite coarse but were suggestive of the basal granules of adjacent ciliated cells. In a few of these cells the granules were scattered throughout the cytoplasm, but in most cases they were massed in the distal portion of the cells, the basal portions being clear and lightly stained (Fig. 23). In others of these nonciliated cuboidal cells the extensions were in the form of long blebs, which had a vesicular or vacuolated appearance (Fig. 24). The cytoplasm in the basal portions of these cells was also relatively clear and lightly stained. The nuclei of all these cuboidal cells presented the same characteristics, being round or irregularly ovoid.

An interpretation of the nature of the bleb-like cytoplasmic projections just described could not be made with certainty. The vacuolated appearance was very indicative of a degenerative phenomenon occurring in the distal portion of the cytoplasm, which would be difficult to understand in view of the normal appearance of the proximal portions and nuclei of these cells.

Epithelial hyperplasia, which was frequently found in our material, occurred in two forms: (1) Hyperplasia principally of the ciliated cells and (2) hyperplasia principally of the goblet cells. In consideration of the first mentioned type, examination of the various specimens indicated first that in some instances there was a very noticeable hypertrophy of the ciliated cells as compared with the most simple form. The columnar cells were considerably taller and the cilia were very pronounced also (Fig. 11). Further increase in the thickness of the epithelium, however, was not accompanied by further cellular hypertrophy but by increase in compactness with crowding and further multiplication, finally producing stratification (as noted by Kistner, 1913) (Figs. 9, 10, 12 and 13). In some cases the epithelial thickness was more than double that described as normal. When the epithelium was stratified the cells lining the free surface were very highly ciliated, forming an almost complete ciliated surface. In some cases numerous young goblet cells, which were identified by their densely staining mucinogen granules, were found lying between the ciliated cells.

In the cases encountered in which the goblet cells were chiefly concerned not only was there great increase in the number of such cells but they were usually much larger than goblet cells of typical epithelia. Ciliated cells were present in these epithelia and were very tall and slender, spreading out at their distal ciliated ends as though attempting to cover the free surface. In most cases the nuclei were found in the upper broadened out portion of the cells. The goblet cells themselves were quite cylindrical in shape except where narrowed down at the free border. The secretion free portion of their cytoplasm was extremely scanty and their nuclei much shrunken and compressed in the basal portion of the cells. It was often impossible to trace extensions of these cells between the prominent basal cells to the basement membrane, suggesting stratification (Figs. 15 and 16).

We were fortunate in securing four specimens from maxillary sinuses which had been subjected to complete resection of mucous membranes shortly before, so that they showed a regenerating type of epithelium. One of these, which had been operated upon just one month previously, possessed an epithelium very similar to that described by McGregor (1931) in a specimen of sixteen days' regeneration.

The cells of the single layer of epithelium found in this month-old mucous membrane varied greatly in size and shape from cuboidal to columnar. Many of the cylindrical cells were clubbed at their free ends, in which case the nucleus was to be found in this enlarged portion. Some of the cuboidal cells would be better described as pyramidal, the base of the pyramid lying on the basement membrane. In a few places cells were found which apparently did not reach the basement membrane, but, due to the great irregularity in cell shape, this could not be positively stated. In no portion did the epithelial thickness approach that of a normal mucosa and in no portion did it appear that the adjacent cells were in complete contiguity with one another. The nuclei of all these cells were conspicuously large in proportion to the cytoplasm and stained so intensely that nuclear details were largely obscured. The cytoplasm, when stained with Delafield's hematoxylin and azur II-eosin, was of a lilac color and quite homogeneous. No evidences of developing cilia or of the development of goblet cells were to be found (Fig. 25).

The connective tissue found in connection with the hyperplastic types of epithelia indicates the presence of varying degrees of inflammation in these membranes. The most abundant infiltration of lymphocytes, macrophages and eosinophiles (in six cases) occurred in connection with hyperplasia of ciliated cells. Where hypertrophy and

hyperplasia of the goblet cells were noted the underlying connective tissue was usually found to be quite densely fibrous, particularly just underneath the basement membrane, and was much less cellular, indicating, possibly, subsidence or chronicity of the infective process.

The connective tissue found in the regenerating membrane was very loose, highly vascular and contained numerous young fibroblasts.

THE FRONTAL SINUS.

Relatively little material from the human frontal sinus was available for our study. In the operative material that we received only scattered portions of epithelium remained. This was a tall pseudostratified ciliated columnar epithelium such as occurred in the maxillary sinuses, except that no goblet cells were found. The amount of epithelium found, however, was so small that it could not be said to be representative. The connective tissue underlying such epithelium was rather dense but gave no other indications of pathologic processes.

In the dog and calf material examined, the epithelium was of a low columnar type with relatively few basal cells to be found along the basement membrane. The same was true of that found in the cat, except that the columnar cells were taller. The cilia were quite long in all three instances but were not as numerous as found in the human material described above. Goblet cells occurred only occasionally (Figs. 1, 3 and 7).

Returning to the human material, we found quite a different appearance in that secured at autopsy. Although the connective tissue was in essentially the same condition as that found in the surgical specimens, the surface epithelium was found to be composed almost exclusively of goblet cells, except in small areas where columnar ciliated cells were comparatively numerous (Fig. 17). Very thin, tall ciliated cells were also to be found occasionally lying between the goblet cells but making up a very minor part of the epithelium. A heavy layer of mucus covered the surface in which were found nuclei and other cellular débris. In one area the superficial cells had sloughed leaving only the basal cells. In another area remnants of goblet cells and their nuclei were found apparently in the process of sloughing away from the basal layer (Fig. 22). These appearances led us to the belief that when these goblet cells had become completely filled with mucin they disintegrated either as the secretion was liberated or immediately thereafter.

THE ETHMOID CELLS.

The epithelium found in this region differed from that found in the maxillary sinus chiefly in the presence of large numbers of

goblet cells. In two cases this feature was not so apparent, the epithelium being composed of a thick layer of ciliated cells with prominent basal cells. Scattered throughout the layer of ciliated cells, however, were numerous young goblet cells, in early formative stages, identified by numerous heavily stained mucin granules in their cytoplasm.

In the other six specimens examined, however, there was a great preponderance of distended goblet cells in the superficial layer. These were so closely pressed together as to appear as long columns of mucus, rather than of typical goblet form. The few ciliated cells found were very tall, and so compressed as to be exceedingly thin. Distally these ciliated cells spread out laterally in order to clothe a larger surface with cilia. In many places the surface cells either had sloughed or were in the process of sloughing off, leaving only the prominent irregular basal cells (Figs. 18, 19, 20 and 21).

The principal evidence of pathologic disturbances found in the connective tissue associated with this epithelium was an infiltration of cells with eosinophilic granulations found in four cases.

Very little similarity was found between the mucous membrane above described and that found in the mucosa of the ethmoid cells in the calf. In the latter case the epithelium was of a columnar ciliated type with a few scattered basal cells and no goblet cells were found.

THE SPHENOID SINUS.

As in the case of the frontal sinus, little human material from this sinus was available for study, being limited to two specimens both secured at autopsy. In neither case were any evidences of pathologic disturbances found, but slight differences in the character of the epithelium in the two cases were encountered. In one instance the superficial ciliated cells were lower than in most of the material we have studied. A few well filled goblet cells were scattered here and there (Fig. 6). In the second specimen the superficial ciliated cells were considerably taller and more compactly arranged, in some places seemingly stratified. Among these cells were found numerous mucin containing cells in various developmental stages leading to goblet cells. These were made the object of special study. Following mucicarmine staining, large numbers of tall, cylindrical cells were seen which contained from a few to many deeply stained granules in the region of the nucleus in the basal portion of the cell. In some such cells the cytoplasm was filled with such granules, the nucleus being pushed into the basal portion of the cells and conforming to the shape of the basal process. In such cases the nuclei were very compact and pycnotic

and much smaller than the nuclei of the ciliated cells. In other cells these granules had become swollen and somewhat vesicular, forming sometimes mucus plugs. The continued evolution and fate of such cells has been seen in material collected from the other sinuses.

DISCUSSION.

In consideration of the various morphologic appearances presented by the epithelia which serve as lining for the paranasal sinuses there seems little doubt that these epithelia possess great adaptive powers to cope with the constant and rapid changes in environment to which they are subjected because of their close relationship to the external environment of the individual. If the environmental insult is at a minimum and the individual not greatly susceptible to upper respiratory disease, we would expect to find the simplest type of epithelium commensurate with the primary motile and protective function served by the respiratory epithelia. This hypothetic epithelium would be composed of a single layer of low columnar or cuboidal cells, all bearing motile cilia, with possibly an occasional undifferentiated cell lying on the basement membrane but not reaching the free surface. We have not been able to find such simple epithelia in the paranasal sinuses of man, but in some of the material collected from the dog, cat and calf, in which nasal diseases are relatively infrequent, epithelia were often found which were very similar to that above described, with the exception that occasional goblet cells were found in between the ciliated cells in some instances (Figs. 1, 2 and 3).

A condition only slightly more complex than this we have often found occurring in the human maxillary and sphenoid sinuses under favorable conditions, and quite frequently in the comparative mammalian material (Figs. 4, 5, 6, 7 and 8). This increase in complexity consisted principally in an increase in height of the columnar ciliated cells and an increase in the number and size of the undifferentiated basal cells. In one specimen of human maxillary sinus it will be noted that no goblet cells were found, which was frequently true of the material from other sources. In all of these specimens the cilia of the surface layer were very long and numerous, and proven, by inspection under incubation temperatures, to be highly motile. The remarkable protective powers of this ciliated border have been amply shown, particularly by recent investigators (Linton, 1930; Proetz, 1929, 1932; Hilding, 1932). The increased height of the columnar cells would give added protection to the underlying tissues and the increase in number of the basal cells, a factor in the greater speed of replacement of sloughing or disintegrating superficial cells.

As the environmental insult becomes more severe, as supposedly occurs under conditions of acute inflammation, it would seem that greater and greater protection of underlying tissues would be called for and that greater and more rapid replacement of lost superficial cells would be necessary. These requirements would be met by increased compactness and thickness of the epithelium so that the number of ciliated cells reaching the surface would be increased and the number of basal cells not reaching to the surface would be markedly increased, such as was frequently found occurring in maxillary sinus membranes surgically removed during attacks of acute sinusitis. In such cases the superficial cells were much increased in height, sometimes to a remarkable degree (Figs. 9, 10, 11 and 12). Such an epithelium was found in one infected maxillary sinus removed from a rabbit. This hyperplastic state was occasionally found to have reached such a degree that the epithelium could almost with certainty be spoken of as stratified columnar ciliated (Figs. 13 and 14).

Two features of additional interest were noted in the hyperplastic epithelia above described. First, in some cases we found that the number of cilia on the surface did not seem to keep pace with the cellular increase so that there was at least a relative decrease in the number of cilia (Fig. 10). Possibly associated with this decrease in cilia and their power to cope with adverse environmental conditions and also possibly associated with environmental changes of such a nature that increased thickness, replaceability and ciliary motility were not suitable protection, we sometimes found in these hyperplastic epithelia evidences of increase in the proportion of goblet cells (Fig. 13). Similar change has been experimentally produced in the nasal epithelium by blocking one nostril (Hilding, 1932). These mucus secreting cells were quite typical in size and content. The possibility of their origin from ciliated cells must be considered in view of the presence of large vacuoles in the distal portion of the cytoplasm in certain ciliated cells of some hypertrophic epithelia (Fig. 11). The spaces or vacuoles might have been filled with mucus, but we were unable to demonstrate this by selective staining. In spite of the fact that the nuclei of such cells appear very vesicular and large, it seems to us that this frothy appearance would be more plausibly considered as a peculiar degenerative phenomenon. In other material we have found more definite early stages of development of goblet cells in the superficial cell layer in that mucin granules were present in variable number in the distal portion of the cytoplasm of certain columnar cells in which cells no cilia were demonstrable.

Under apparently more severe or long standing adverse conditions we have found increasing proportions of goblet cells present in the surface layer of epithelium. In many cases by far the majority of the lumen surfaces of the sinuses were occupied by huge goblet cells. The tendency of the remaining ciliated cells to spread out on the lumen surface makes it appear that the surface is still covered by cilia. Such an epithelium was found in two surgical specimens of maxillary sinuses (Figs. 15 and 16).

As the proportion of goblet cells increased, we have found instances in which the ciliated cells were present as islands, all other surface being occupied by goblet cells of tremendous size (Figs. 17, 19, 20 and 21). Under such circumstances the undifferentiated basal cells with their large vesicular nuclei and relatively homogeneous cytoplasm become very prominent.

In the epithelia containing such great numbers of hypertrophic goblet cells we found considerable evidence suggesting the essentially degenerative nature of these cells, as intimated by Ross (1932) from tissue culture studies, in contradistinction to the findings of Florey and Webb (1931) in their studies of the goblet cells in the colon. In many cases, when the goblet cells had become well filled with secretion, the nucleus was seen to have become a pycnotic mass and the basal portion of the cytoplasm immediately around it to have shrunken, the two elements indistinguishably combined into a pyramidal mass which stained brilliantly with acid fuchsin (Figs. 17 and 18). As the cytoplasm of the mucus secreting cells became shrunken, the basal part often was noted to have lost contact with the underlying tissue so that the epithelium could be spoken of as truly stratified (Fig. 17).

As a further step in the disintegrative process, we have found areas in the epithelial surfaces in which it was evident that the goblet cells had completely disintegrated, leaving cytoplasmic and nuclear remnants in a mass of mucus. Extremely vague cell outlines could sometimes be seen, but it seemed obvious that the goblet cells were in the process of disintegrating and sloughing. In other portions of the epithelium we have found areas of considerable extent in which the columnar superficial cells had disappeared leaving only the basal layer of cells covering the connective tissue (Figs. 21 and 22).

In some regions, where the epithelium was thus reduced in thickness, we found some interesting evidences of histogenetic processes. In a portion of one specimen, a single layer of low cuboidal cells was

present, many of which bore a few cilia on the free surface (Fig. 5). In other regions of the same specimen we found, in the peripheral cytoplasm of such cuboidal cells, numerous densely stained granules, the significance of which has not been exactly determined but which might be considered as centrosomal granules concerned in the production of cilia (Fig. 23). In still other regions of this specimen, where the epithelium was reduced to a single layer, some of the cells presented a very peculiar appearance in that the basal portion of their cytoplasm in the region of the nuclei seemed to be quite normal in appearance, whereas the cytoplasm of the peripheral portion of the same cells was quite vacuolated and frothy in appearance, seemingly undergoing a form of degeneration (Fig. 24). In between such cells were scattered cuboidal cells which were ciliated. At present we are unable to offer adequate explanation of the peculiar appearance of these cells.

The underlying connective tissue in those cases in which such an abundance of goblet cells were present was extremely dense, and, in some instances, so dense that it appeared as a hyalin mass, giving an intense connective tissue staining reaction. This appearance in the connective tissue is, we believe, indicative of either a chronicity of reaction or the beginning subsidence of an acute inflammatory reaction. In our experience this appearance has always been associated with an hyperplastic epithelium which contained a very high proportion of goblet cells.

As has been noted before, during the time that we have been collecting the materials for this study, we were fortunate enough to secure a sinus membrane from an individual in which a complete membrane resection had been done only a short time previously (twenty-four days). Regeneration of both the epithelium and connective tissue was apparently proceeding rapidly. The epithelial cells, which did not completely cover the underlying tissue, were irregular but tended toward a columnar shape (Fig. 25). They were quite undifferentiated, appearing to be quite similar to the undifferentiated basal cells of the more complex types we have described. These cells of the regenerating mucosa were very similar to those described by Gorham and Bacher (1930) and by McGregor (1931), after sixteen days of regeneration. The connective tissue was also of a regenerative type, with numerous fibroblastic cells present. This regenerating mucous membrane could not easily be compared with those which have been described by Coates and Ersner (1930) and Semenov and Kistner (1930), because of differences in the time allowed for regeneration and the treatment employed. In a specimen of three weeks'

regeneration shown by Gorham and Bacher, no epithelium was present, and in one of one month's duration the epithelium was ciliated.

Such a regenerative undifferentiated type of epithelium as we have illustrated and described would possess the potentialities of differentiation into any of the varieties of epithelia which have been described, depending upon the conditions encountered within the sinus as differentiation proceeded.

CONCLUSIONS.

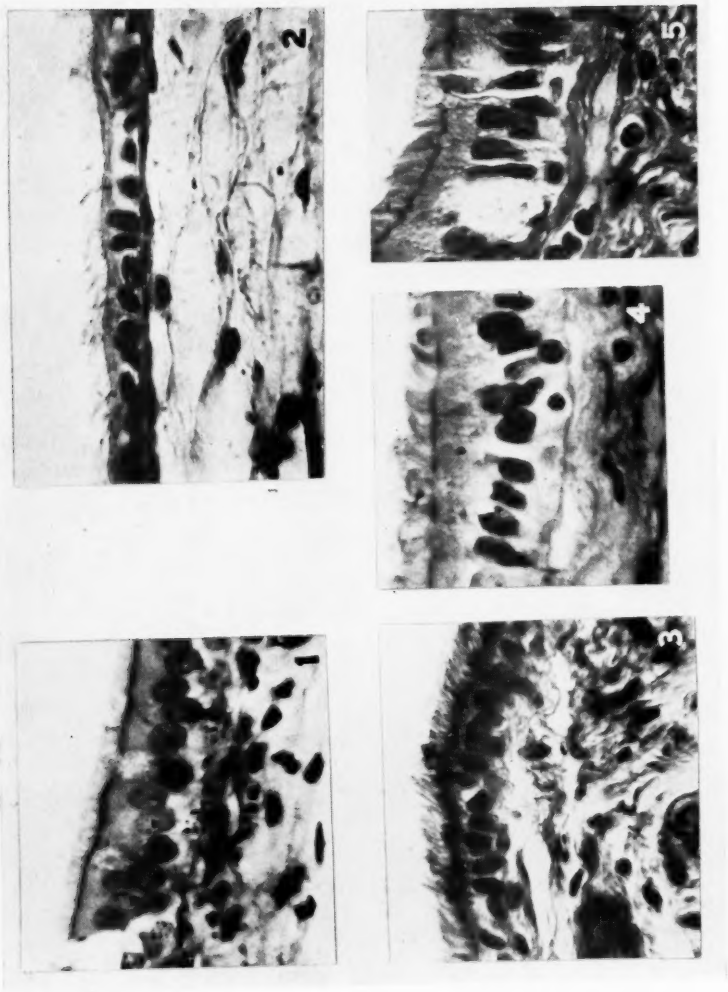
In conclusion, it might be said that the simplest type of epithelium which was found as lining of the paranasal sinuses was a practically simple columnar ciliated epithelium with only occasional basal cells present. In the type found to any extent in the human sinuses and those of the domestic animals, the relative number of undifferentiated basal cells was somewhat greater, so that the epithelium was pseudostratified, either columnar or cuboidal (some of comparative material). Practically all the superficial cells in these cases were equipped with long and numerous cilia. Either no or relatively few goblet cells were seen in these cases. Such an epithelium was most frequently encountered in the maxillary and sphenoid sinuses and was found associated with a relatively thin underlying connective tissue stroma.

Under conditions of an acute irritation, the basal cells, through their more rapid growth and differentiation, evidently brought about a hyperplasia of the epithelium, so that it becomes thicker, the cells more closely packed and the superficial cells in addition more heavily ciliated. In these epithelia the proportion of goblet cells has not noticeably increased. Such hyperplasia apparently sometimes continues until a true stratified columnar epithelium is present. This hyperplastic type of epithelium was always associated with an underlying connective tissue which had become infiltrated with considerable numbers of macrophages, polymorphonuclear leucocytes and lymphocytes, giving evidence of an acute inflammatory process. This type of epithelium was most often found as lining of the maxillary sinuses.

In other instances in which epithelial hyperplasia was evident, considerable numbers of large goblet cells were found in the superficial layer. In some cases the ciliated cells remaining were greatly compressed but tended to spread out at the lumen border in order to clothe a larger surface with cilia. In many cases, however, practically the entire lumen surface was lined by these very large goblet

cells. The origin of these cells is considered to be principally the undifferentiated basal cells, but some appearances seem to indicate that occasionally ciliated cells may be thus changed also. Evidences have been presented pointing to the essentially degenerative nature of these mucin secreting cells terminating in their disintegration as their secretion is liberated. This type of epithelium was found associated with a very dense underlying connective tissue, in some cases so dense as to be nearly hyaline, which indicated that either a chronic irritative condition was present or that the tissue was in the reparative stages following an acute inflammatory process. Epithelia in which such remarkable numbers of goblet cells were present (epithelial mucoid degeneration) were invariably found in material from the human ethmoid cells and in the few specimens obtained from the frontal sinuses (human).

Regeneration of the epithelium apparently occurs from the irregularly cuboidal undifferentiated basal cells, which are not involved in any degenerative sloughing process unless environmental conditions are extremely severe and, even then, only a part of them are affected. These cells, we believe, possess the potentialities of forming either ciliated or goblet cells, depending upon the environmental stimulus present while they are differentiating.


2
5
4
1
3

EXPLANATION OF FIGURES.

PLATE I.

Fig. 1. Frontal sinus, dog. This illustrates a rather low pseudostratified columnar ciliated epithelium resting upon a fairly compact connective tissue. Basal cells and goblet cells are scattered. The basal granules of the numerous cilia are very prominent. Heidenhain's iron hematoxylin. Photo x960.

Fig. 2. Maxillary sinus, calf. This shows a flattened or cuboidal pseudostratified (or stratified ciliated) epithelium. The basal cells are much flattened upon the very loose underlying connective tissue. A relatively small number of cilia are to be found on the free surface. Delafield's hematoxylin and azure II-eosin. Photo x960.

Fig. 3. Frontal sinus, calf. The superficial cells are somewhat taller and the cilia much more numerous than in the preceding illustration from the same individual. Delafield's hematoxylin and azure II-eosin. Photo x960.

Fig. 4. Maxillary sinus, human. In this instance the epithelium is seen to consist principally of relatively tall columnar ciliated cells. A very few basal cells may be seen between basal processes of the columnar cells. Note the absence of goblet cells. Mayer's haemalum and eosin. Photo x960.

Fig. 5. Maxillary sinus, human. Similar to the preceding illustration, the columnar cells being somewhat taller. The basal granules of the cells are very apparent. A portion of one goblet cell appears in the section. Delafield's hematoxylin and Mallory's aniline blue connective tissue stain. Photo x960.

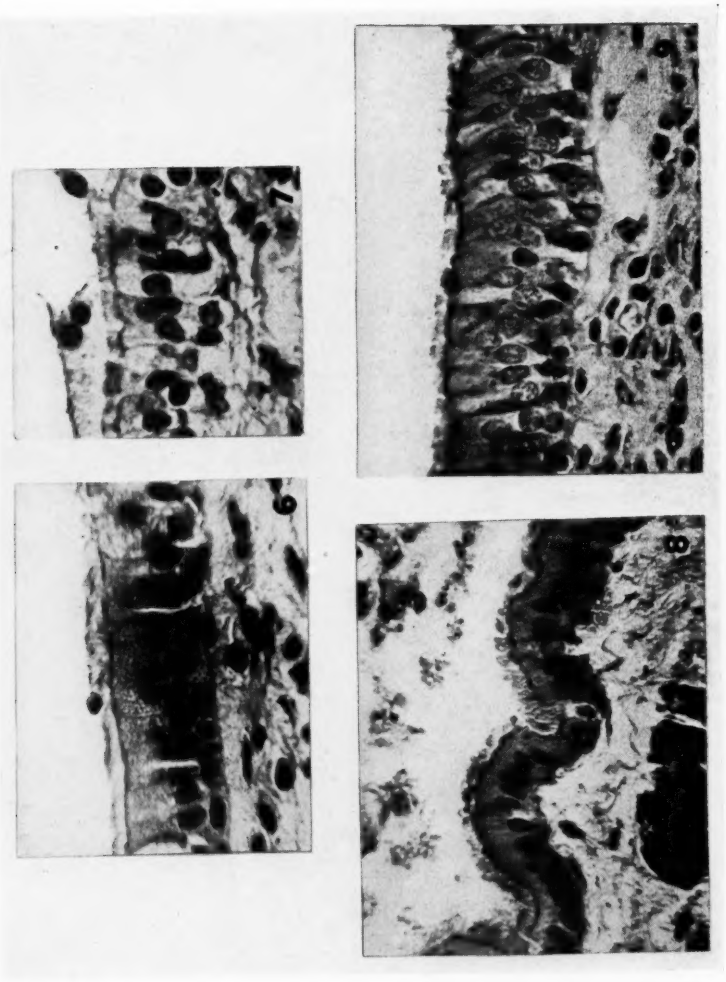


PLATE II.

Fig. 6. Sphenoid sinus, human. An apparently normal mucosa. The columnar cells are relatively short, goblet cells few in number and only one in this field, basal cells scattered and covering a loose connective tissue. Delafield's hematoxylin and azure II-eosin. Photo x960.

Fig. 7. Frontal sinus, cat. Similar to the preceding illustration, with less cytoplasmic structure apparent and no goblet cells. Delafield's hematoxylin and azure II-eosin. Photo x960.

Fig. 8. Maxillary sinus, rabbit. Normal mucosa with no goblet cells found. The basal cells flattened out along the basement membrane. Delafield's hematoxylin and azure II-eosin. Photo x720.

Fig. 9. Maxillary sinus, human. A taller, more compact epithelium than any of the preceding. Basal cells are more numerous. No goblet cells apparent. The epithelium rests on a normal tunica propria. Delafield's hematoxylin and azure II-eosin. Photo x720.

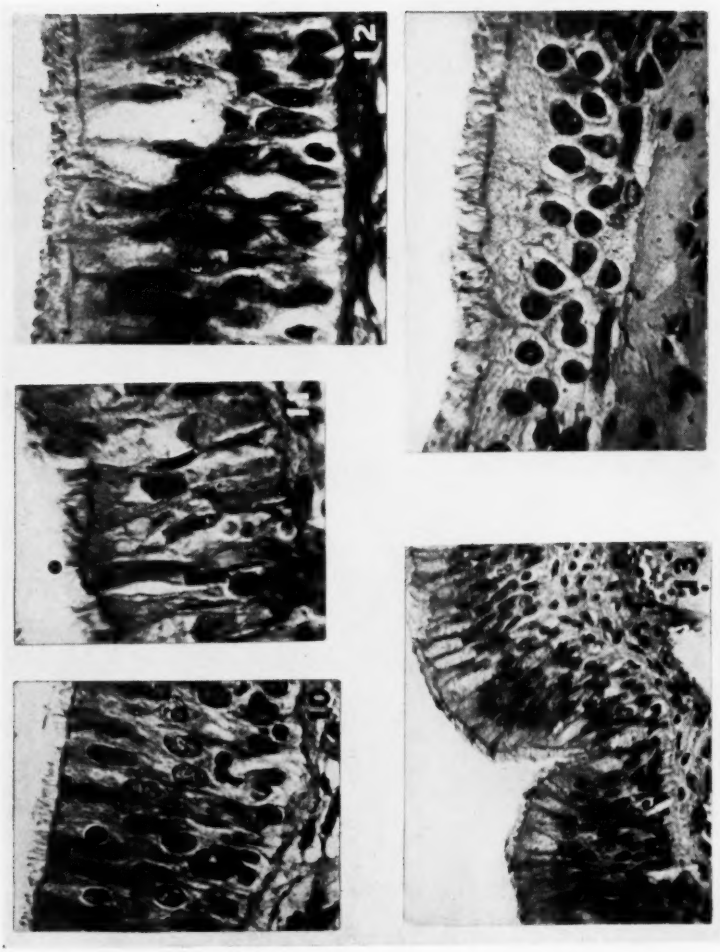

12
11
10

PLATE III.

Fig. 10. Maxillary sinus, human. A more hyperplastic epithelium than the preceding. Basal cells have increased in number. The number of cilia is much reduced. The lighter stained cells are developing goblet cells. Delafield's hematoxylin and azure II-eosin. Photo x960.

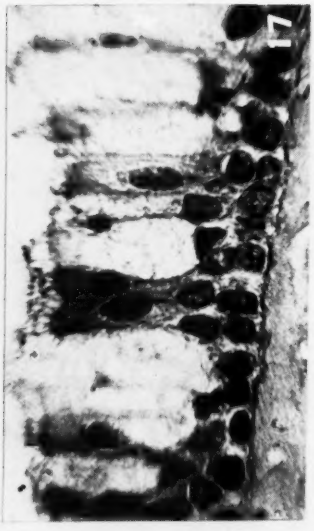
Fig. 11. Maxillary sinus, human. The epithelium is somewhat hypertrophic. The vacuoles appearing in some of the cells are considered indicative of degeneration. Delafield's hematoxylin and Mallory's aniline blue connective tissue stain. Photo x960.

Fig. 12. Maxillary sinus, human. Marked hyperplasia and hypertrophy of the ciliated cells. Goblet cells, one with a brilliantly fuchsin stained basal mass, are seen. Note the character of the connective tissue. Weigert's iron hematoxylin and picrofuchsin. Photo x960.

Fig. 13. Maxillary sinus, human. More marked epithelial hyperplasia. Goblet cells are fairly numerous. Quite evident change is seen in the connective tissue. Delafield's hematoxylin and Mallory's aniline blue connective tissue stain. Photo x440.

Fig. 14. Maxillary sinus, cat. In this case the epithelium is arranged in a stratified manner, the columnar superficial cells are bearing cilia. The underlying connective tissue is quite dense. Delafield's hematoxylin and Mallory's aniline blue connective tissue stain. Photo x960.


16


17

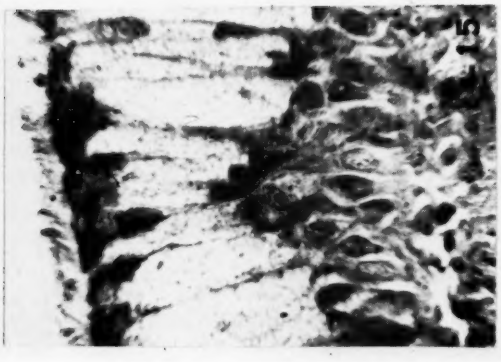


PLATE IV.

Fig. 15. Maxillary sinus, human. A very hyperplastic epithelium, with goblet cells predominating. The Ciliated cells spread out at their free borders to cover a large part of the surface. Heidenhain's iron hematoxylin and Mallory's aniline blue connective tissue stain. Photo x960.

Fig. 16. Maxillary sinus, human. In this instance, as in Fig. 15, most of the hypertrophied superficial cells are goblet cells. The cytoplasm of the remaining very slender ciliated cells can be seen between adjacent goblet cells. Note the numerous basal cells here and in the preceding figure. Weigert's iron hematoxylin and picrofuchsin. Photo x960.

Fig. 17. Frontal sinus, human. Again an hyperplastic hypertrophic epithelium with predominating goblet cells. Ciliated cells appear as small groups. The basal cells are still more prominent than in preceding figures, forming apparently a complete layer. The cytoplasmic and nuclear remnants of some of the largest goblet cells are well indicated here. Delafield's hematoxylin and Mallory's aniline blue connective tissue stain. Photo x960.

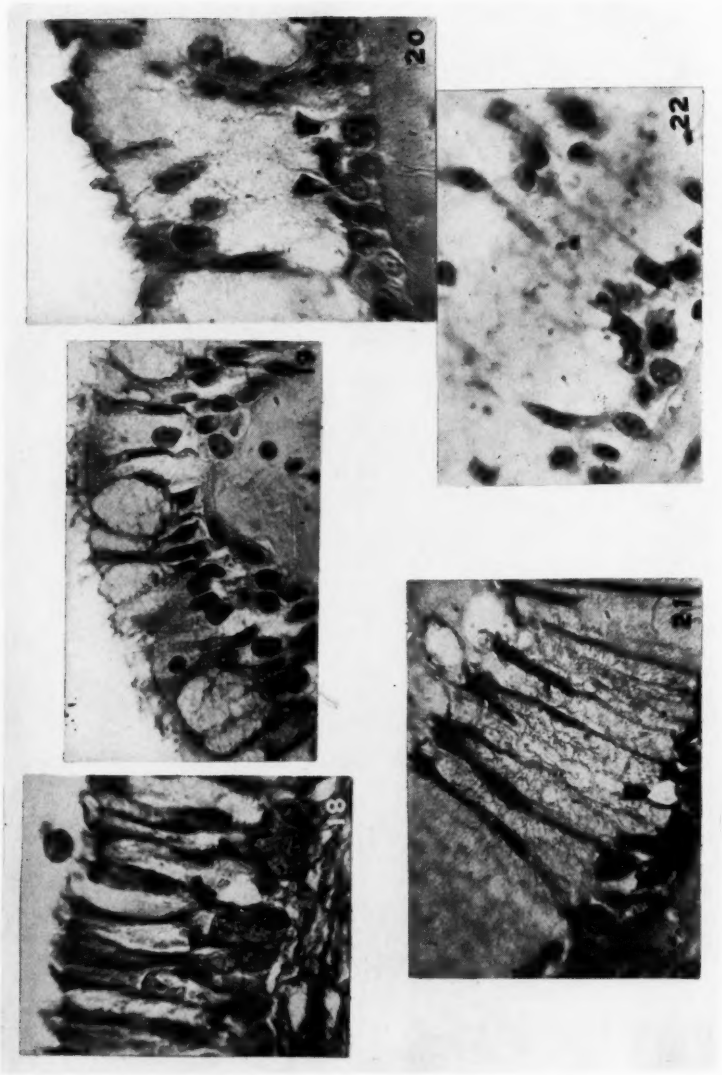

20
22
18
21

PLATE V.

Fig. 18. Ethmoid sinus, human. In this case goblet cells are not quite so prominent. Many of them appear to be empty of secretion. The shrunken basal processes of some of the cells which stained so intensely with acid fuchsin are well shown. The different basal cells are very prominent. Delafield's hematoxylin and Mallory's aniline blue connective tissue stain. Photo x960.

Fig. 19. Ethmoid sinus, human. Numerous well filled goblet cells are to be seen in the superficial epithelial layer with very slender ciliated cells lying between them. Cilia are not so numerous as in previous specimens. Delafield's hematoxylin and azure II-eosin. Photo x960.

Fig. 20. Ethmoid sinus, human. This shows a hyperplastic epithelium, the superficial layer of which consists almost exclusively of very large goblet cells, the remaining cytoplasm and nuclei of such cells being shown as deeply staining pyramidal shaped masses just above the basal cell layer. This latter is almost complete. Delafield's hematoxylin and Mallory's aniline blue connective tissue stain. Photo x960.

Fig. 21. Ethmoid sinus, human. In this epithelium the accumulation of mucus in the superficial cells is still more marked and the degenerative nature of the process quite apparent. At the left the superficial layer seems to have completely disintegrated leaving the basal cells covered with mucus. Delafield's hematoxylin and Mallory's aniline blue connective tissue stain. Photo x960.

Fig. 22. Frontal sinus, human. Here the superficial cells are in a disrupted state with a very few cells attached to the basal layer by slender filaments and with other nuclei and cellular debris lying free in the mucus. The basal cells remain and form a practically continuous layer. Delafield's hematoxylin and azure II-eosin. Photo x960.

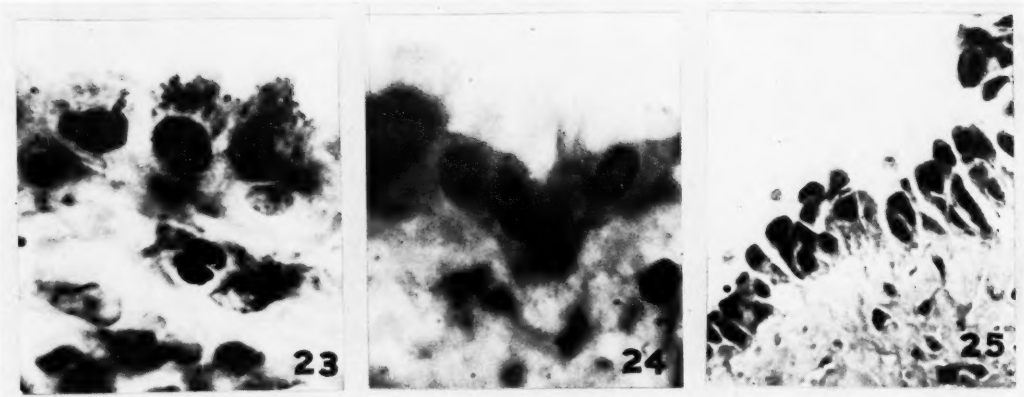


PLATE VI.

Fig. 23. Maxillary sinus. human. The very low superficial cells present give marked evidence of differentiation activity. Note the densely stained cytoplasmic granules collected near the free border of these cells. Delafield's hematoxylin and azure II-eosin. Photo x2400.

Fig. 24. Maxillary sinus, human. Another portion of the same specimen, showing similar low superficial cells, some bearing a few long cilia and others possessing bleblike protuberances which appear as degenerated portions of the cells. Delafield's hematoxylin and azure II-eosin. Photo x2400.

Fig. 25. Maxillary sinus, human. This mucosa was removed from a patient in which a complete resection of the membrane had been done twenty-four days previously. The undifferentiated cells of the re-generating epithelium are very irregular, the cytoplasm relatively scanty and the nuclei so deeply staining that detail cannot be determined. Delafield's hematoxylin and azure II-eosin. Photo x960.

REFERENCES.

Bast, T. H.: Maxillary Sinus of the Dog with Special References to Certain New Structures, Probably Sensory in Nature. Am. J. Anat., 33:449, 1924.

Coates, G. M., and Ersner, M. S.: Regeneration of the Mucous Membrane of the Frontal Sinus After Its Surgical Removal (in the dog). Arch. of Otolaryng., 12:642, 1930.

Florey, H., and Webb, R. A.: Mucous Secretion in Acute Experimental Inflammation of the Colon and Other Mucous Membranes of the Cat: Histologic Changes. Brit. J. Exp. Path., 12:286, 1931.

Gorham, C. B., and Bacher, J. A.: Regeneration of the Human Maxillary Antral Lining. Arch. of Otolaryng., 11:763, 1930.

Hilding, Anderson: The Physiology of Drainage of Nasal Mucosa. II. Cilia and Mucin in the Mechanical Defense of the Nasal Mucosa: A Motion Picture Demonstration. ANNALS OF OTOLOGY, RHINOLOGY AND LARYNGOLOGY, 42:52, 1932.

Hilding, Anderson: Experimental Surgery of the Nose and Sinuses. I. Changes in the Morphology of Epithelium Following Variations in Ventilation. Arch. of Otolaryng., 16:9, 1932.

Kistner, F. B.: Histopathology and Bacteriology of Sinusitis. Arch. of Otolaryng., 13:225, 1931.

Linton, C. S.: A Comparative Study of the Bacterial Flora in Clinically Normal Sinuses. ANNALS OF OTOLOGY, RHINOLOGY AND LARYNGOLOGY, 39:779, 1930.

McGregor, G. W.: Further Proof of the Regeneration of Mucous Membrane in the Human Antrum. Arch. of Otolaryng., 14:309, 1931.

Nemours, P. R.: Studies on the Accessory Nasal Sinuses: The Comparative Morphology of the Nasal Cavities of Reptiles and Birds. ANNALS OF OTOLOGY, RHINOLOGY AND LARYNGOLOGY, 39:1086, 1930.

Proetz, A. W.: Physics and Physiology of Sinuses. ANNALS OF OTOLOGY, RHINOLOGY AND LARYNGOLOGY, 38:963, 1929.

Schaeffer, J. P.: Nose and Olfactory Organ. P. Blakiston's Sons & Co., Philadelphia, 1919.

Schaeffer, J. P.: In Special Cytology. P. B. Hoeber, Inc., New York, 1928.

Schafer, E. A.: Textbook of Microscopic Anatomy (Quain's Anatomy, vol. 2, pt. I, 11th edition). Longmans, Green & Co., London, 1912.

Semenov, H., and Kistner, F. B.: Repair in the Paranasal Sinuses in Man Following Removal of the Mucous Membrane Lining. Proc. of Soc. of Exp. Biol. and Med., 27:322, 1930.

Skillern, R. H.: The Accessory Sinuses of the Nose. J. B. Lippincott Co., Philadelphia, 1913.

Turner, A. L.: Accessory Sinuses of the Nose. Wm. Green & Sons, Edinburgh, 1901.

Ross, P. J.: A Study of Nasal Mucous Membrane in Hanging-drop Tissue Culture. Trans. of the 37th annual meeting of Am. Acad. of Ophth. and Otolaryng., p. 432, 1932.

LXXIX.

PSEUDO-ABSCESS OF THE BRAIN: INTRACRANIAL DISEASE DURING OTITIS MEDIA SIMULATING ENCEPHALIC ABSCESS.*

J. M. Nielsen, M. D.,

AND

Cyril B. Courville, M. D.,

Los Angeles.

When in the course of otitis media or mastoiditis the usual and benign symptomatology is interrupted by signs of exacerbation or complication the otologist thinks of intracranial extension of the inflammatory process. The most frequent complication within the cranial cavity is extradural abscess, after which follow with about equal frequency sinus phlebitis and leptomeningitis. Actual abscess formation within the substance of the brain is not common, yet its recognition is important inasmuch as its proper treatment leads to more uniformly favorable results than does the best treatment of the much more frequent leptomeningitis.

When an abscess of the brain presents characteristic symptoms the diagnosis is not difficult. The unilateral headache, vomiting, slow cerebration, stupor, aphasia if abscess is on the major side, hemianopsia, hemiparesis, bradycardia, subnormal temperature, leucocytosis, mild meningeal symptoms, or cerebellar symptoms with choking of the discs if the abscess is in the posterior fossa, make a definite picture. If it is not typical clinically and yet it is the only intracranial complication of the otitis, the diagnosis may be verified by the process of needling. A real difficulty presents itself when other intracranial complications of otitis media are present simultaneously. The purpose of this communication is to point out, from the standpoint of the neurologist who is attempting to diagnose, but not treat, a number of clinical and pathologic entities which simulate abscess of the brain so closely that at times they defy identification.

*From the Departments of Neurology of the University of Southern California and the College of Medical Evangelists and their Services at the Los Angeles County General Hospital.

Adson[1] was the first to use the term "pseudo-abscess" with reference to conditions simulating abscess of the brain, although the term pseudotumor had been used for some time. He presented histories of three cases. In the first the patient developed contralateral facial paralysis, choked discs, and percussion tenderness over the right temporal region in the course of otitis media. The leucocyte count remained elevated. Needling of the temporal lobe gave negative results but the patient recovered. The second patient, three weeks after scarlet fever, entered with right hemiparesis and motor aphasia, and developed partial right homonymous hemianopsia. Exploration was similarly negative. There was a slight residual hemiparesis and the patient developed petit mal attacks. In the third case the patient developed some astereognosis, aphasia, and bradycardia, the pulse reaching a low point of 50 per minute. This time Adson did not explore but the patient nevertheless recovered.

Symonds[2] and Romantzeff[3] published simultaneously three years later (the first in England, the second in Russia) similar cases, both citing Adson's work. They found eyeground changes, drowsiness, mental aberration, hemianopsia, pathologic reflexes, aphasia, convulsions, not all in the same case but in combinations justifying a tentative diagnosis of abscess of the temporal lobe or cerebellum. Five of the three patients were subjected to exploration; in Romantzeff's cases exploration was carried out on the cerebellum as well as the temporal lobe with negative results. In one of Symond's cases the patient refused operation but recovered equally well. Romantzeff was of the opinion that operation benefited the patients.

However, a search of the literature shows that, while the term pseudo-abscess had not been used, the syndromes described above had been recognized many years earlier. F. Voss[4] credits Oppenheim[20] with having published a statement that sufferers from encephalitis relatively frequently also had a recent or old otitis media. This he had noticed in going over the literature. In a clinical demonstration before the Berlin Gessellschaft für Psychiatrie und Nervenkrankheiten, December 11, 1899, Oppenheim was said to have presented five cases. The clinical picture was that of high fever, slow pulse, motor aphasia, jerking of the opposite arm and face or paralysis in the same distribution. All patients recovered except one (a chlorotic woman), on whom the attending physician insisted on operation.

There are further contributions from many other sources. F. Voss himself presented valuable cases with autopsy or operative findings. He believed the pathologic anatomy to be that of hemorrhagic encephalitis, merging in some cases into abscess formation, and

Yerger[5] and Grinker and Stone[6] have reported somewhat similar cases. The fatal outcome in the latter group shows that the favorable reports of Adson, Symonds and Romantzeff are not always experienced and that cases vary a great deal. Borries[7] has gone into detail on the subject of nonsuppurative otogenic encephalitis and believes that the two types of pathologic anatomy, i. e., suppurative and nonsuppurative encephalitis, are quite different. He dwells especially on the pseudo-abscess syndrome. Cairns,[20] Alexander,[8] O. Voss,[9] Maier,[10] Wischnitz,[11] Marquard,[12] Westergaard,[13] and Key-Aberg[14] have all reported cases of the same general type.

In our experience, these cases are common rather than rare. Of course the variety of cases which may with justification be diagnosed otogenic abscess of the brain depends to a large extent upon one's experience. In some of the above recorded cases we feel that, with the information given in the abstracts, one should not be justified in exploring for abscess. Yet we have seen a number of cases in the last three years in which prior to our study we should have sanctioned operation, but in which we now would be very skeptical of the diagnosis.

CLASSIFICATION OF CASES.

Cases of pseudo-abscess may be classified into certain groups to facilitate organization.

I. Cases of disease of the brain without suppuration and without thrombosis of the lateral sinus. Two subgroups.
   a. Cases in adults; intracranial pressure a prominent symptom.
   b. Cases in young children presenting a syndrome frequently transient, with convulsions or paralysis.

II. Cases associated with sinus phlebitis.

III. Cases of certain lesions of the posterior cranial fossa, as tumor, basilar meningitis, or ependymitis.

IV. Cases of otogenic leptomeningitis.

V. Miscellaneous cases, including multiple emboli, thrombosis of cerebellar artery, syphilis, etc., occurring in the presence of otitis media.

Group 1. Cases of disease of the brain without suppuration and without thrombosis of the lateral sinus. Two subgroups.

a. Cases in adults; intracranial pressure a prominent symptom.

REPORT OF CASE.

CASE 1.—*Chronic otitis media, mastoiditis, possible sinus phlebitis, choking of the discs, subnormal temperature, bradycardia, vomiting, sclerosing petrositis, con-*

striction of fields of vision, marked hysteria, recovery. Dorothy S., age 24, was admitted December 2, 1932, with a complicated setting. Her mother had died at 62 years of diabetes and cerebral thrombosis. She had one sister living, also suffering from diabetes, and there were other instances of diabetes among parents' siblings.

The patient herself had all the usual childhood diseases and also influenza twice, diphtheria once, tonsillectomy and adenoidectomy at 12, and appendectomy with peritonitis at the age of 13. She had also had a right phrenicotomy at 21, as well as a pelvic operation at the same age and later alcohol injection of the left phrenic nerve for persistent hiccough.

She had been married at 16, had had six pregnancies with only two living children, of which one had died of mastoiditis. Wassermann reactions had been negative.

Patient was distinctly obese. She first came to the Los Angeles County General Hospital August 2, 1932, with acute exacerbation of her old otitis media and right mastoiditis. White cell count was 16,200 per cu. mm. Roentgenogram showed sclerotic mastoiditis right. A simple mastoidectomy was done and the patient was discharged August 31 with slight discharge still present.

She was readmitted two weeks later because of continued pain, profuse discharge from the ear, increasing mastoid tenderness, headaches and hiccoughs. X-rays showed no change. The hiccough was recognized as hysterical and a history was then obtained to the effect that it had been present during previous illness. (The phrenicotomy and injection had then been done.) We found the hiccough occurred sixty-six times per minute. There were no signs of intracranial complications of otitis media.

We saw her again October 31, 1932, because of rapid myoclonic jerkings of head, neck and nearly entire body, but by compensatory synchronous jerkings in the opposite direction (tonic neck reflexes) the eyes were held stationary and a fundus examination was easily performed.

There was no change in the eyegrounds. Deep reflexes were increased in the right extremities. There were no pathologic reflexes present. We believed that the patient had some organic irritation of the brain plus hysteria. On November 3 the mastoid was reopened and shortly thereafter the hiccough ceased.

Patient entered again December 2, 1932, because of pain, which had appeared two days before in the right side of the head, face, jaw and neck, and because of chills the previous night. There was still discharge from the right ear, while the left was negative. Both fundi showed hyperemia and blurring of the disc margins but this was slight.

We saw her December 12, but found measurable elevation of both optic discs (1½ to 2 D). On the left the veins were quite tortuous. Deep reflexes did not lead to a localizing diagnosis. Patient was having chills and fever, and we believed she had a thrombus of the lateral sinus. At operation Dr. Semenov did not verify this but did a jugular ligation. The chills and fever ceased immediately.

December 15 patient had pain in the right eye; Babinski and Oppenheim reflexes were present and the deep reflexes were increased on the right.

On December 20 Dr. Werden examined the patient and because of a temperature of 96.2, pulse rate below 70 per minute, choked discs increasing in severity, motor weakness of the left leg, increase in left knee jerk, and positive Babinski and Chaddock left, made a diagnosis of abscess of right temporal lobe. This indeed seemed justified.

On December 22 we found the choking of the discs still greater (4½ to 6 D), but could not verify the reflex changes and the pulse did not remain slow. A roentgenogram showed increased density of the right petrous bone.

A stormy month now ensued. There were the most bizarre findings at various times, the reflexes shifting in activity from side to side. Pathologic reflexes were never consistently present, and patient was at all times hysterical. Vomiting became so marked that patient was unable to keep even water on her stomach. Intramuscular fluids were given for more than a week. Severe headache, pain in the right eye and bouts of chills and fever appeared. Fields of vision showed a partial bitemporal hemianopsia.

The choking of the discs gradually subsided, fever disappeared and it was decided to discharge the patient for psychic effect. The needles were withdrawn from her thighs and she was told to eat normally because fluids into the muscles were no longer necessary. To our own surprise, the patient arose, dressed and walked out of the hospital. She was asked to return for observation in a week, and on the appointed day appeared after having driven twelve miles through heavy traffic. The choking was less. Nystagmus was still present but patient was feeling quite well. There was a partial left homonymous hemianopsia. She continued to return for several months and all symptoms disappeared. She has remained well to date (more than a year). Our final diagnosis was petrositis.

We confess this case was extremely difficult to evaluate. Petrositis may cause cerebral or cerebellar symptoms or both. Had the patient maintained a subnormal temperature and had we then known where to explore we should have favored needling. Removal of the hysterical symptoms (vomiting, weakness of the left lower limb, prostration) by the psychotherapeutic discharge from the hospital greatly clarified the situation. The visual field defects may also have been hysterical in nature but these remained for a time.

b. Cases in young children presenting a syndrome, frequently transient, with convulsions or paralysis.

Subgroup b is a type of case in which the authors have been particularly interested and of which a series of twenty-three has been reported.[15] We have been unable to find the syndrome described in the literature as such, yet there are occasional cases reported under a variety of titles. Kennedy[23] recently reported two cases as illustrations of cerebral edema due to otitis media. The clinical picture varies somewhat, but in all twenty-three cases there was one manifestation in common—that of a focal cerebral lesion with signs of motor irritation or paralysis, and disturbance of consciousness occurring in the course of otitis media. The great majority of the patients were in the second and third years of life (ages one and two). These cases cannot be distinguished clinically from those of abscess of the brain except by the course of the disease, as we have found in other cases appearing at first like these but in which abscess subsequently developed. The following case is illustrative:

REPORT OF CASE.

CASE 2.—*Acute otitis media, mastoiditis, loss of consciousness, general and focal convulsions, left hemiplegia, choked discs, negative exploration, residual hemiparesis.* Tomasa B., a Mexican girl of seven years, who had been well prior to May 29, 1932, was taken ill on that date with fever, anorexia, headache, pain in both ears without discharge, gastro-intestinal symptoms, and listlessness. This state persisted with exacerbations and remissions for two weeks when she became nauseated and vomited three times in one day.

She was admitted to the Los Angeles County General Hospital June 12 at 1:00 a. m., unconscious and in a convulsion which had begun on the left side. Left jacksonian seizures occurred repeatedly during the first few hours in the hospital and these left the patient with a left hemiplegia, the only peculiarity of which was that the forearm was flexed strongly on the arm, the face and lower extremity being relatively little affected.

On examination the patient was found to be extremely irritable; there was some rigidity of the neck and a Kernig sign bilaterally in complete conformity with a spinal fluid pressure of 420 mm. of spinal fluid containing 65 white blood cells and 35 red cells per c. mm. A bilateral otitis media was found and mastoiditis was shown by roentgenogram. The neurologic examination gave what one would expect—i. e., absence of deep reflexes on the affected side and pathologic reflexes immediately, but increased reflexes after a few days. The patient did not talk when conscious. At the time of admission the eyegrounds showed only a slight blurring of the nasal margins, but this increased during the next two weeks until an elevation of the discs to 1.5 D. left and 2 D. right was present. The white blood count was 22,440 per c. mm. at time of entrance. Mastoidectomy was done.

The course was not smooth. Rises of temperature were infrequent but a low temperature or slow pulse never occurred. Thrombosis of the lateral sinus was excluded. In six weeks she still had a blood count of 13,000, but the spinal fluid was normal. The patient gradually resumed talking, first answering in monosyllables. The improvement in the hemiplegia was slow and we abandoned hope of complete recovery. When she did begin to walk there was a tendency to flexor spasm in the lower limb. The upper extremity slowly straightened fairly well but remained weak. It was our impression that so long as the patient was clinically improving there was no haste about draining a temporal lobe abscess if present.

After three months improvement ceased. We felt that it was not safe to discharge the patient without exploration in view of the fact that cerebral abscesses are known to be almost symptomless in the latent stage, only to cause the death of the patient later. Accordingly, on September 20th, one of us (C. B. C.), under ether anesthesia, explored the right temporal and also the parietal lobes. Exposure of the parietal area disclosed a diffuse atrophic change in the cortex with excess of fluid in the subarachnoid space. A second opening above and behind the ear revealed a normal cortex but puncture of the temporal lobe met with some diffuse resistance, suggesting a regional gliosis. A photograph of this patient is shown in Fig. 1.

This case has been reported before in an exposition of this syndrome,[15] but is selected again for presentation here because of the residual paralysis which is permanent record of the lesion. Yet we wish to emphasize that the majority of these patients do not have residuals, many recovering after very transient episodes of convulsions or paralysis.

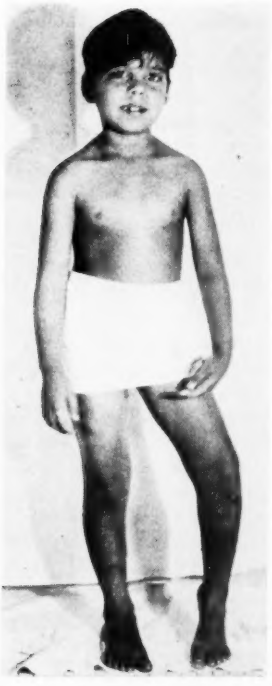


Fig. 1. Photograph of patient in Case 2, showing contractures and left hemiplegia as residuals of nonsuppurative involvement of the brain.

We cannot be dogmatic concerning the pathologic anatomy in these cases, but there is evidence to show that from the middle ear extension occurs by way of the superficial veins of the cortex, in most cases toward the parietal lobe. When, therefore, we see a child of one or two years enter with otitis media and jacksonian convulsions, usually of an upper extremity, conjugate deviation of the head and eyes, loss of consciousness, and who a day or two later shows a hemiplegia, we watch closely for developments. He may be well the next day; he may improve slowly, or he may die; yet we do not advise exploration for abscess. At that stage exploration cannot help the patient. If he later develops bradycardia, choked discs and stupor, we consider needling for abscess. In a great many cases the patient recovers completely without needling or in spite of it.

Group II. Cases associated with sinus phlebitis.

The second type of pseudo-abscess of the brain to be presented is that associated with thrombosis of the lateral sinus. The literature

on this subject is especially rich, and a review of it makes it evident to us that in every case of thrombosis of the lateral sinus one must be exceedingly careful not to conclude without clear-cut evidence that abscess formation within the brain has also occurred. Klestadt[27] points out that the operation on the sinus may in some cases be the cause of the cerebral symptoms as it was in the case of Skeyde from his clinic. In such cases there must be damage to the inner wall of the sinus. Klestadt's cases presented bradycardia, stupor, vomiting, headache, slow cerebration, subnormal temperature and choked discs, making exploration seem indicated. Salinger[16] reported two cases, in one of which jacksonian seizures and in the other hemiplegia with conjugate deviation of head and eyes was present. Hoffmann[17] reported a case of right sided facial paralysis of central type and bradycardia, for which reason the brain was explored but with negative results. In Herlinger's[18] case there were aphasia, paresthesias, somnolence, pathologic reflexes, slight choking of the discs and nystagmus. Exploration several times remained entirely negative and recovery occurred. The patient of Bodechtel and Richter[19] came to autopsy revealing a sclerosis of the parietal cortex of the opposite side. Mann[20] reported a case of left sided otogenic sinus thrombosis in which after jugular ligation, motor aphasia and paralysis of the right arm appeared. Trephine over Broca's area showed fibrinous deposits on the pia and a gelatinous character of the cortex. The patient recovered after four months. Neubauer's[21] case resembled much that of Bodechtel and Richter in that the right sided sinus thrombosis gave rise to left sided symptoms of aphasia and right hemiplegia. The author believed the left parietal lobe to have been affected perhaps by a toxin. Blau[22] gathered from the literature 162 cases of sinus thrombosis in which eyeground changes had been found in 112, or 69 per cent. This shows that that particular symptom should not be taken to indicate abscess of the brain in preference to lateral sinus thrombosis. A case illustrative of this group follows:

REPORT OF CASE.

CASE 3.—*Otitis media, ipsolateral headache, loss of weight, vomiting, stupor, irrationality, amnesic aphasia, right homonymous hemianopsia, mildly choked discs, bradycardia, subnormal temperature, leucocytosis, thrombosis of left lateral sinus.* Henry C., aged 17, who had been suffering with osteomyelitis of the right scapula for five months, was taken February 24, 1932, with painful left otitis media without discharge. During the last week of April he began to have left sided headache distinctly more severe in the early morning, pain in the left eye, loss of appetite and body weight. By the middle of May vomiting began and on May 19 he became drowsy. On May 20th he slept all day, and on May 21st he became irrational and had short periods of deep stupor for which reason he was admitted to the Los Angeles County General Hospital.

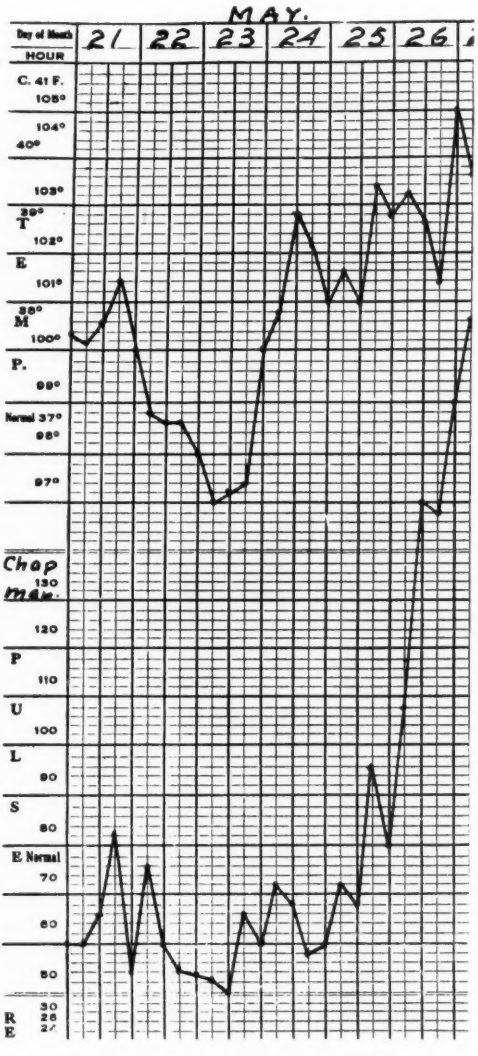


Fig. 2. Pulse and Temperature chart in Case 3. The subnormal temperature and bradycardia are striking but they fail to persist. (Thrombosis of the lateral sinus.)

On entrance he was pale and listless, had difficulty in talking, complained of severe pain in the left frontal parietal regions. The left pupil was slightly larger, and right homonymous hemianopsia was determined by Dr. Werden. Both optic discs were slightly elevated on the nasal margins. There was a tendency to conjugate deviation of head and eyes to the left. There was also weakness of the right face on emotional expression, not on volition. The deep reflexes were greater on the right where a suggestive Babinski sign was also elicited. Dr. C. W. Rand found the patient unable to name a pencil or watch (amnesic aphasia). A charting of the visual fields confirmed the impression of right homonymous hemianopsia. Temperature was 100.4 F., pulse rate 60 per min.

Blood count showed 18,500 leucocytes per c. mm., of which 89 per cent were polymorphs. Spinal fluid contained only 3 cells per c. mm., but it was under a pressure of 350 mm. of water.

The patient was immediately taken to surgery, where the temporal lobe was punctured in various directions, but in vain.

Fig. 2 shows the relations of the pulse and temperature the following two days. With a temperature down to 97 F. and pulse rate below 60 it was difficult indeed to understand why an abscess of the brain should not be present. Slow cerebration was prominent, as were headache, nausea, vomiting, rigidity of the neck, stupor, and the hemianopsia persisted. The aphasia increased so that such expressions as "all right," "what's the matter," and a few other expletives constituted his entire speech. A partial Wernicke's hemianopic pupillary inaction sign was found, indicating involvement of the left optic tract. Pathologic reflexes became definite. Elevation of the optic discs increased, and on May 25th the patient never spoke except once to say "water." Leucocytosis increased to 32,800 per c. mm., and pneumococci appeared in the spinal fluid. Surgery was again tried, the brain being punctured further posteriorly and inward but again in vain. He finally developed right hemiplegia, became involuntary and incontinent and died May 26th.

At autopsy a thrombosis of the left lateral sinus was found. There was no abscess of the brain.

Group III. Cases of certain lesions of the posterior cranial fossa, as tumor, basilar meningitis or ependymitis.

The third type of pseudo-abscess is that in which the patient has a lesion of the posterior fossa as shown by choked discs, meningeal signs, severe headache, etc., and one must of necessity consider cerebellar abscess. If the patient has suffered from otitis media in the course of which the symptoms appear, the differential diagnosis may be difficult indeed. A subacute or chronic basilar meningitis, a cerebellar tumor invading the fourth ventricle, or ependymitis may simulate cerebellar abscess very closely. A recent case in point seen by us follows:

REPORT OF CASE.

CASE 4.—*Chronic otitis media, acute severe headache, bilateral proptosis, ophthalmoplegia externa, vomiting, choked discs, bradycardia, leucocytosis, cerebellar tumor felt by needling, all symptoms independent of otitis.* Dorothy G., aged 15, had suffered for years with chronic recurrent otitis media, the last exacerbation of

which had occurred a year before. Three weeks prior to entrance into the Los Angeles County General Hospital on the Contagion Service she was taken with headache which became progressively worse. A private practitioner made a diagnosis of influenza. Just before admission she lapsed into coma.

When examined neurologically she had bilateral marked proptosis with ophthalmoplegia externa bilateralis and injection of the conjunctivæ. She was vomiting, had bilateral choking of the discs and bradycardia. White count was 12,000 per c. mm. Our impression was thrombosis of the cavernous sinus. However, the acute symptoms subsided, the patient cleared up mentally, both mastoids were found to be clear roentgenologically and the patient took on the appearance of one with a cerebellar lesion on the left. Both abducens nerves became paralyzed, the choking increased, marked ataxia of the left upper extremity developed. Spinal puncture gave clear fluid under increased pressure with 4 cells per c. mm. Slow pulse and low temperature appeared. After a week of observation Dr. Patterson was persuaded to needle the left cerebellar lobe for abscess with a plan in advance to proceed as for tumor if no abscess was encountered. A large tumor was found by needling in the left cerebellar lobe.

In this case the history of chronic otitis media with acute coma, proptosis and ophthalmoplegia externa, rigidity of the neck, Kernig sign, and leucocytosis all pointed to intracranial extension of the purulent process, yet all the symptoms were due to an acute episode of a chronic cerebellar lesion, probably tumor.

Group IV. Cases of otogenic leptomeningitis.

Patients with otogenic leptomeningitis may present symptoms closely simulating cerebral or cerebellar abscess. This has been pointed out by nearly all writers on the subject. A particularly striking case has come to our attention.

REPORT OF CASE.

CASE 5.—*Chronic otitis media, mastoiditis, mastoidectomy, choking of the discs, bradycardia, ligation of jugular, ataxia and weakness of right arm, finally hemiplegia, negative exploration of cerebrum and cerebellum, due finally due to meningitis.* E. W. M., a boy of 19, gave a history of having had recurrent otitis media with drainage for two years, the last attack occurring two months prior to admission. This subsided but recurred after five weeks with vertigo and severe pain in the right ear, about the right eye, and in the zygomatic region. After a week of this, paralysis of the right face developed.

The patient was admitted with facial paralysis, throbbing pain in the right ear but no diplopia or blurred vision. The vessels of the right disc were slightly engorged and tortuous, the cup small. On the left the cup was obliterated, the veins still more full, the margins elevated. The left pupil was smaller, deep reflexes increased on the left, left Gordon and Oppenheim and suggestive Babinski reflexes were present. There was no nausea, vomiting or chill. The white blood count was10,000 cells per c. m., polymorphs, 75 per cent.

After a few days the blood count rose to 17,000 cells per c. mm., and the spinal fluid then showed 26 cells per c. mm. The temperature chart (Fig. 3) shows how

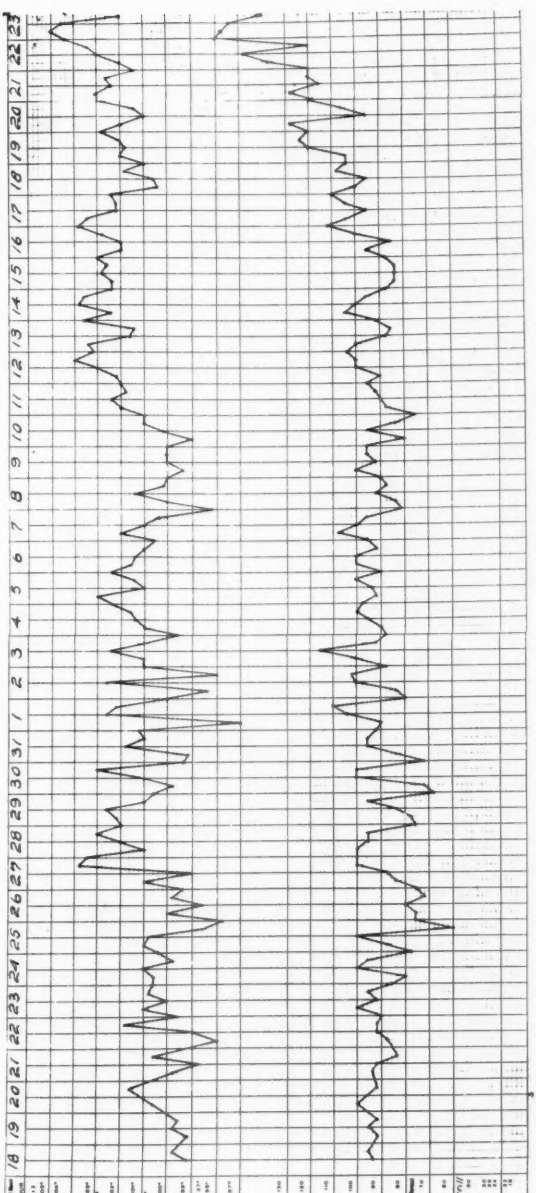


Fig. 3. Pulse and temperature chart in Case 5. Subnormal temperature and bradycardia are due to meningits.

suggestive of encephalic abscess the curves were up to the 26th, when the temperature was 97.8 F., and the pulse 60 per min. The pulse was peculiarly slow through most of the course, but considerable fever developed after that point.

Dr. Detling and his staff proceeded with mastoidectomy immediately and found cholesteatoma, erosion of the dural plate, exposure of the facial nerve, and purulent involvement of the horizontal semicircular canal. All this was treated without delay, but by November 11th, the patient was delirious at intervals, the right pupil was greater, further elevation of the optic disc on the left developed; there were some incoordination of the eye movements at times, vertical nystagmus on looking upward, marked atonia and some ataxia of the right arm and meningeal symptoms and signs. The deep reflexes were greater on the left; Babinski, Chaddock, Gordon and Oppenheim reflexes appeared. The jugular was then ligated and the sinus packed. The cerebellum and right temporal lobe were explored by one of us (C. B. C.), but with negative results. The course continued downward and the patient died after having developed right hemiplegia. Autopsy showed septic basilar meningitis but no abscess of the cerebrum or cerebellum.

Group V. Miscellaneous cases, including multiple emboli, thrombosis of cerebellar artery, syphilis, etc., occurring in the presence of otitis media.

Rare disease combinations may be gathered for collective brief consideration. Benesi and Brunner[24] have reported a case in which in the presence of mastoiditis there was an unrecognized heart condition from which multiple emboli entered the brain, creating a clinical picture of abscess of the temporal lobe, chiefly aphasia and nystagmus. Esch[28] has reported a somewhat similar case. Heike and Lewy[25] have reported a case in which a woman of sixty years, during an attack of otitis media, developed cerebellar symptoms. Operation was delayed, but finally undertaken as an emergency measure. The patient then died and at autopsy it was determined that the symptoms were due to a thrombosis of the posterior inferior cerebellar artery. Such errors as the above are never to be avoided entirely.

Syphilis presents an ever-present problem. Whenever a positive Wassermann reaction is found on routine examination in the presence of otitis media and symptoms of cerebral involvement occur, the temptation is strong to recommend antiluetic treatment and dismiss the suspicion of abscess of the brain. The following case is illustrative:

REPORT OF CASE.

CASE 6.—*Syphilis, cranial injury, bleeding ear, earache, swelling about mastoid, fundus changes, diplopia, subnormal temperature, bradycardia, leucocytosis, no abscess of the brain.* Mrs. M. L., aged 44, had been well prior to March 28, 1933, when she fell and struck her head on a cement floor. There was no loss of consciousness, but she was dazed and spent nine days in the hospital. On the second day in hospital she had pain in both ears and bled from the right, and there was said to

have been swelling below the mastoid region. Then followed a period of six weeks at home during which she had nervous tremors and occasional headaches. About the middle of June she took to her bed, was dizzy and nauseated but did not vomit. October 13th she again developed earache and swelling in the region of the right mastoid. After a week she became irrational following a series of chills, was dizzy and felt as though she rotated toward the right. On admission to the Los Angeles County General Hospital, she had a rise of temperature to 101 and was drowsy and nauseated.

At the time of neurologic examination she complained of headache and there was tenderness to percussion over the right side of the head as well as to pressure over the right mastoid. Prominence of the veins of scalp and eyelids was noted. Pupils were small and reacted sluggishgly to light and the eyegrounds showed engorgement and tortuosity of the veins. There was a little nystagmus and diplopia in the primary position. Deep reflexes were sluggish throughout. Babinski, Gordon, Chaddock, Oppenheim reflexes were present bilaterally and marked dysmetria was found on the right. Moderate meningeal signs were present. Roentgenogram showed sclerotic mastoids, the right more involved. The Wassermann and Kahn tests were both four plus. Her temperature became subnormal and the pulse reached 60 per minute at times. White blood count was 16,000 per c. mm.; spinal fluid negative.

Here was a neat problem in diagnosis. Subdural hematoma, otitis media with extradural abscess, and cerebral abscess were all to be considered in addition to the syphilis. She was first treated for lues but made no progress. We then felt that mastoidectomy was indicated for diagnosis, but the staff of the Ear, Nose and Throat Department did not consider this necessary. On our insistence a simple mastoidectomy was done. There was no pus anywhere. The patient was then treated for syphilis. Subsequent events have shown that cerebral abscess is in all probability not present, as there is recession rather than advance in symptoms.

We have learned that it is necessary to consider two simultaneous diseases in such cases. We advise treating the syphilis while closely observing for brain abscess and then exploring if the symptoms of abscess still increase.

In view of the difficulties pointed out, how are we then to determine when in the course of otitis media an abscess of the temporal lobe or cerebellum has developed and needs exploration? The answer is—not by one visit or even one careful examination but by repeated systematic follow-up examinations, plus a knowledge of what is apt to develop in such a case. To this is added diagnostic surgery, which we do not hesitate to advise. When the eardrums have been opened but symptoms continue we study the mastoids. When the mastoids have been drained, the dural and sinus plate removed, and the lateral sinus studied, one has determined the presence or absence of extradural abscess and lateral sinus thrombosis. These procedures will, if carefully carried out, not only remove infection and hasten recovery but will place the examiner in a position to know whether extension has occurred. This greatly simplifies the diagnosis. If the patient is known not to have extradural abscess, subdural abscess or menin-

gitis, yet symptoms and signs of intracranial complications are still present, exploration for abscess of the brain is certainly justified. In competent hands this, carefully done, does the patient little or no harm, and is the only positive proof of freedom from abscess of the brain. It must constantly be borne in mind that abscess of the brain is not a surgical condition as soon as it develops. There is ample time for study. Ordinarily a week or two may be allowed and during this time daily study will certainly indicate the course of the disease.

The authors are greatly indebted to Doctors Detling and Homme, heads of the Nose and Throat Services at the General Hospital, and their associates for their gracious co-operation and help in permitting us to follow the cases on their services.

BIBLIOGRAPHY.

1. Adson, A. W.: Pseudobrain Abscess. S. Clin. North America, 4:503-512. (Apr.), 1924.

2. Symonds, C. P.: Some Points in the Diagnosis and Localization of Brain Abscess. J. Laryng. and Otol., 42:440-448. (July), 1927.

3. Romantzeff, N.: Pseudo-abscess. Russk. Klin., 8:67-71. (July), 1927.

4. Voss, F. (Riga.) Drei Fälle von Encephalitis im Anschluss an Otitis media. Ztschr. f. Ohrenheilk., 41:223-233. 1902.

5. Yerger, C. F.: Acute Toxic Meningo-encephalitis of Otorhinogenic Origin. Arch. Otolaryng., 1:190. (Feb.), 1925.

6. Grinker, R. R., and Stone, T. T.: Acute Toxic Encephalitis in Childhood: A Clinicopathologic Study of Thirteen Cases. Arch. Neurol. and Psychiat., 20:244-274. (Aug.), 1928.

7. Borries, G. V. Th.: L'encephalite otogène hémmorrhagique. Rev. de laryng., 53:49-72, (Jan.) 1932.

8. Alexander, G.: Meningo-encephalitis mit trübem, sterilem Lumbalpunktat. Exploration des Schläfenlappens. Monatschr. f. Ohrenh., 60:365-368 (Apr.), 1926.

9. Voss, O.: Otitis media und Encephalitis. Ztschr. f. Hals-, Nasen-, u. Ohrenheilk., 21:596-608. Festschrift Otto Körner. 1928.

10. Maier, M.: Erfahrungen über den otitischen Hirnabszess. Arch. f. Ohrenh., 95:163-238. (June), 1914.

11. Wischnitz: Meeting of the Berlin Otological Society, Dec. 6, 1912. Reported in Arch. f. Ohrenh., 91:164, 1913.

12. Marquard, Kurt: Ueber ungewöhnlich lokalizierte Encephalitisformen nach Grippe. Mit einem Beitrag uber das Symptom der Adiadochokinese. Arch. f. Psychiat., 67:84-104. 1923.

13. Westergaard, A.: Fractionierte lumbal Punktion bei otogenen Meningitiden, Kopenhagen. 1931. Cited by Borries.

14. Key-Aberg: Contribution à l'étude de l'encephalite otogène. Acta Otolaryng., X 75. 1926.

15. Courville, Cyril B., and Nielsen, J. M.: Concerning Certain Cerebral Manifestations Following Acute Otitis Media in Infants and Young Children. To appear in Am. J. Dis. Child.

16. Salinger, S.: Generalized Clonic Spasms, Hemiparesis and Coma: The Result of a Lateral Sinus Thrombosis. Laryngoscope, 33:27-30. (Jan.), 1933.

17. Hoffmann, R.: Ausgedehnte, nicht infizierte Thrombose mehrere Hirnsinus und der Jugularis in Folge einer Operationsverletzung des Sinus transversus. Heilung. Ztschr. f. Ohrenh., 30:17-35. 1897.

18. Herlinger, I.: Klinische Beobachtung. Ein Fall ausgedehnter Thrombose des Sinus sigmoideus und transversus mit Schläfenlappenabszesssymptomen und abnorm grosser Einmündung einer Vene (wahrscheinlich V. Cerebri) in den Sinus transversus. Monatschr. f. Ohrenh., 66:999-1000. (Aug.), 1932.

19. Bodechtel, G., und Richter, H.: Ueber Scheitellappensymptome bei otogener Thrombophlebitis der Gegenseite. Ztschr. f. Hals-, Nasen-, und Ohrenh., 32:505-516. (Mar.), 1933.

20. Mann: Motorische Aphasia mit gleichzeitiger Lähmung des rechten Armes nach linkseitiger otitischer Sinusthrombose. Monatschr. f. Ohrenh., 64:1207-1212. 1930.

21. Neubauer, A.: Nach akuter mittel Ohrentzündung (a) aufgetretene Sinusthrombose mit Aphasia und Hemiplagie. Jahrb. f. Kinderheilk., 54:361-364. 1924. (Abstract in Zentralb. f. Hals-, Nasen-, u. Ohrenh., 6:83. 1925.

22. Blau, L.: Zur Lehre von den otogenen intrakraniellen Erkrankungen. Gehirnabszess, Sinusthrombose, Meningitis. Beitr. z. Anat., Physiol., Path. u. Therap. d. Ohres., 10:86-156. 1918.

23. Kennedy, F.: Acute Intracranial Edema Complicating Otitis Media. Reported before the New York Neurological Society and New York Academy of Medicine, Section of Neurology and Psychiatry. Nov. 10, 1931. Reported in Arch. Neurol. and Psychiat., 27:1497-1498. (June), 1932.

24. Benesi, O., und Brunner, H.: Multiple Hirnerweichungen unter dem Bilde eines otogenen Schläfenlappenabszesses. Monatschr. f. Ohrenh., 55:714-736. 1921. Jubiläumscheft zum 50 Jahre des Bestandes der Allgemeinen Poliklinik in Wien.

25. Heike, H., and Lewy, F. H.: Klinik und Pathologie eines atypischen Falles von Verschluss der Art. cerebelli post. inf. Monatschr. f. Psychiat. u. Neurol., 36:26-41. 1914.

26. Oppenheim, H.: Die Encephalitis und der Hirnabscess. Alfred Hölder, Wien. 1897.

27. Klestadt, W.: Zerebrale Symptomenklomplexe bei otogener Sinusphlebitis. Ztschr. f. Laryng., Rhin., Otol., XIII, 83-105. 1925.

28. Esch, A.: Embolisher Erweichungsherd oder otogener Hirnabscess im linken Schläfenlappen. Klin. Wchnschr., 1:781-783. Apr. 15), 1922.

29. Cairns, H.: Brain Abscess. J. Laryng. and Otol., 45:385-397. (June), 1930.

SYMPOSIUM ON THE CONSERVATIVE TREATMENT OF THE NOSE, THROAT AND EAR.*

LXXX.

CONSERVATIVE TREATMENT OF THE NOSE AND THE ACCESSORY SINUSES.

W. E. GROVE, M. D.,

MILWAUKEE.

The purpose of my discussion of this subject is to outline the principles of conservative treatment of the nose and sinuses. It is not my idea to create any argument between the proponents of conservative and radical surgery which both have a very definite place in nasal therapeutics.

The abnormal, pathologic conditions of the nose and sinuses which call for any sort of treatment can, with very few exceptions, be placed in four general groups: malformations of the structural framework of the nose, infections, allergic conditions and neoplasms. Only the treatment of infections and of allergic conditions will logically fall within the scope of this discussion because the treatment for malformations of the nose and for neoplasms is purely surgical and frequently radically so.

In the treatment of infections of the nose and sinuses, one must consider, first of all, the defense mechanism of the organism and then how that defense mechanism can be assisted and stimulated by treatment.

Referring to the nose and accessory sinuses, the first line of defense against infection consists in the ciliary activity of the nasal and sinus mucosa and the mucinous blanket layer which overlies the ciliated epithelium.[1 2 3] To these must be added, though of minor importance, the action of air currents within the nose during inspiration and expiration, and the variations of air pressure within the sinuses during respiration.

*From the scientific program of the American Academy of Ophthalmology and Laryngology, September 13, 1934.

The second line of defense consists of the phagocytic action of macrophages and microphages; the action of opsonins, lysins and agglutinins and the reactions of natural immunity, both general and local.[4 5]

Of the many influences which tend to undermine the natural resistance of the mucosa, the most important are: diet, physiologic effects, allergy and trauma. By physiologic effects we refer to chilling, ventilation, chemical irritation, metabolic fluctuations and obstruction to drainage. Linton[5] regards all of these factors as important because any one of them may be the deciding factor in causing a break in the defense of the mucosa. Schmidt[6] has called attention to the action of cold on the skin and mucosa of the upper respiratory tract in causing a prolonged contraction of the blood vessels of skin and mucosa. Because of this prolonged vasoconstriction the defense forces can no longer be easily mobilized and the ever present bacteria find a favorable field for development.

Our treatment of infection of the nose and sinuses, whether conservative or radical, must be designed to assist the natural defenses of the mucosa and restore its function to normal. The normally functioning mucosa of the upper respiratory tract with its mucinous layer, its ciliary action and its reticulo-endothelial elements is marvelously efficient in protecting the individual against infection.

Under ordinary circumstances the offending bacteria are engulfed in the mucinous blanket and are propelled, with varying degrees of rapidity, toward the pharynx, esophagus and stomach, where they are destroyed. When, because of obstructed drainage or physiologic effects, such as persistent chilling, bad ventilation, chemical irritation and metabolic disturbances, the action of the ciliated mucosa is interfered with, mass action of the invading organism takes place, the epithelium is penetrated, the sinuses are invaded. Leucocytic exudation to combat the invading organism results in free secretion or pus in the nose and sinus cavities to such an extent that stagnation occurs, the movement of the mucinous blanket is still further hindered and a vicious circle initiated. Our defense mechanism has broken down.

Our efforts at this point must be concentrated upon the reduction of swollen mucosa, the removal of stagnant secretions and the stimulation of the mucosa to resume its normal activity. This can be accomplished by applications and packs of various kinds to shrink the sinus ostia, by irrigations of various sinuses to remove stagnant secretion and by other efforts to increase the proper ventilation of the sinuses.

In this connection the experiments of Lierle and Moore[7] are illuminating. These investigators experimented with the action of various drugs on the ciliary activity of the mucosa in man, dogs and guinea pigs, and came to the following conclusions:

1. Tap water and distilled water, when applied to the mucosa of the upper respiratory tract, causes a slowing of the ciliary beat.

2. Three per cent ephedrin hydrochlorid is not detrimental to ciliary activity but at times slightly increases it.

3. Five per cent cocain hydrochlorid is not detrimental to ciliary activity, but 10 and 20 per cent solutions produce definite slowing with good recovery.

4. Mild silver protein solutions of 5, 10 and 20 per cent produce an initial speeding of ciliary activity with a subsequent slowing, probably due to the water solvent.

5. One-half per cent eucalyptol is not detrimental.

6. One-half per cent and to a greater degree, 1 per cent, menthol has a mildly depressing effect.

7. One per cent thymol, ½ and 1 per cent eucalyptol are definitely detrimental to ciliary activity in the order named.

8. A 1-1,000 epinephrin hydrochlorid, a 2 per cent zinc sulphate and a 2 per cent mercurochrome solution, in the order named, are definitely detrimental to ciliary activity.

9. One-half per cent silver nitrate solution is immediately and fatally detrimental to ciliary activity and in no instance was it again possible to start ciliary beating after its application.

With the above facts in mind, we will probably make some changes in our applications of medication to the nasal and sinus mucosa. We can increase natural drainage and stimulate ciliary activity by the application of some of the above mentioned drugs to the nasal mucosa and more particularly to the sinus ostia. Medication, more particularly ephedrin solution, can be directly introduced into the sinus cavities by the displacement method as advocated by Proetz.[8] Whether the displacement method is any additional help in diagnosis seems to be a mooted question, but Proetz[8], Frazee[9] and others have rather definitely shown that it is of much benefit in conservative treatment of nasal and sinus infections.

Where our efforts to assist the mucosa are blocked by stagnation within the sinuses, these stagnant secretions can be removed by normal salt solution irrigations. Such irrigations can be carried out on all the sinuses except those of the ethmoid capsule. In many cases the normal ostia can be catheterized. Puncture of the antrum has been

performed since the very beginnings of rhinology. Watson-Williams[10] has popularized the puncture of the sphenoid, and Van Alyea[11] has recently advocated the puncture of the floor of the frontal sinus where the normal opening could not be probed.

Drainage and ventilation of the antrum can be still further improved by a window resection of the inferior meatal wall, that of the frontal sinus by infraction of the middle turbinate or the removal of the operculum, and that of the sphenoid by removal of its anterior wall.

When the above outlined measures have failed, when the sinus infection has passed from an acute or subacute into a chronic condition, when the lining mucosa has been replaced by a pyogenic membrane with enormous increase in the connective tissue elements, then and then only has our conservative treatment failed to benefit the patient and more radical measures must be resorted to.

Diet, climate and sunshine play a large rôle in the prevention and cure of sinus infections. The fact that colds are practically unknown in the arctic and relatively rare in the semi-arid belt, but very common in regions where wide variations of temperature and humidity prevail deserves consideration from the standpoint both of prophylaxis and treatment. Shea[12] says, "The older my sinus patient happens to be, the quicker I send him searching for the sun." Certain it is that sinus patients who cannot find comfort in the climates of rapid temperature and moisture changes are much benefited by living in regions like Southern Florida, Southern California and on the eastern slopes of the Rockies. If sunshine is unavailable, Shea recommends daily ultraviolet radiation to the entire body and within the nose. I heartily concur in the first half of this recommendation because the action of ultraviolet radiation, particularly from the sun itself, is a very potent factor in assisting the body to auto-immunization against respiratory infections. I have, however, never seen any benefit from the direct application of ultraviolet therapy to the nasal mucosa itself.

With respect to diet, Jarvis[13] believes that the absence of fat in the diet of young individuals tends to increase lymphoid tissue in the throat and that the lack of uncooked vegetables in individuals from twenty to thirty will increase the catarrhal discharges from nasal and sinus membranes. Cody,[14] working on the effects of vitamin deficiency diets, maintains that vitamin A in the diet is necessary to maintain the nutrition of the nasal, aural and tracheal mucosa. Prophylactically, vitamin A seems to increase the resistance to upper respiratory infections in children. Therapeutically, its chief value is to improve

the nutrition of the nasal and aural mucous membranes in acute and chronic infections.

According to Cody, a diet, deficient in vitamin B, is identified with a nasal syndrome consisting, essentially, of a postnasal discharge and the posterior tips of the middle turbinates are smooth, white, moist and slightly thickened.

Vaccines.—The normal sera or plasma have some antibacterial activity. (Harkness.[4]) When infection takes place auto-immunization begins and the cells of the reticulo-endothelial system begin to throw out antibodies to combat the bacterial antigens (Fenton and Larsell[15]) in addition to increasing enormously the numbers of the micro- and macro-phages. Clinically, we can definitely assist this auto-immunization process by diet, hydrotherapy, heliotherapy and vaccines, both specific and nonspecific.

The best results from specific vaccine therapy are obtained from autogenous vaccines used over a considerable time. However, for more immediate action some form of nonspecific protein therapy may be employed. For this purpose we can use parenteral injections of such substances as lactogen, boiled milk, typhoid vaccines and omnadin. (Shea[12] and Mithoefer.[16]) In my own hands omnadin has been very effective.

In the prevention of ordinary seasonal colds I have found very successful the use of certain stock vaccines, worked up gradually to an immunizing dose, which dose is repeated once or twice monthly during the winter months.

Before leaving the subject of vaccines I must call attention to the subject of local immunity as produced by the specific nonfilterable antivirus solutions as worked out by Besredka.[17] Besredka and his co-workers have shown, both in vitro and in vivo, that bacteria cannot live and develop in the presence of their own homologous antiviruses, either in the skin, in the eye or in the peritoneal cavity. Kolmer,[18] in cases of chronic bronchitis and bronchiectasis, made intrabronchial injections of specific antiviruses, and believes that, in addition to the bacteristatic and bactericidal action for its own specific organism, the antivirus owes its therapeutic properties to stimulation of both microphagic (polymorphonuclear) and macrophagic (reticulo-endothelial) phagocytosis, together with a local antibody production. Mulso,[19] using a stock antivirus, had marked success in rapidly healing the mouth lesions of agranulocytosis.

If all these facts be true, it would appear profitable to inquire into the possible therapeutic benefit to be derived from intrasinus applications of specific antiviruses.

Allergy.—A constantly increasing number of nasal and sinus conditions which formerly masqueraded under the designations of vasomotor rhinitis, hyperesthetic rhinitis, recurring colds, polyposis and the like have more recently been recognized as manifestations of allergic states and conditions. Efficient treatment depends upon correct diagnosis, and this diagnosis must identify the offending allergen or allergens. In arriving at this diagnosis a most careful and painstaking history is of prime importance, as Mullin,[20] Hansel,[21] Rowe[22] and others have pointed out. This history must be a general inquiry into the patient's present and past illnesses, habits and idiosyncrasies. It must also include a careful inquiry into the medical history of his immediate ancestors. This history is then supplemented by physical examination, by cytologic examinations of nasal smears, by differential blood counts and by skin sensitization scratch tests. It has, however, been pointed out by various workers in this field that skin tests are frequently negative in cases where true allergy exists. Therefore, too much reliance should not be placed on these tests alone. The elimination diets of Vaughn[23] are frequently helpful in making a diagnosis.

The diagnosis once made, the treatment will entail the close cooperation of rhinologist, internist and allergist. Any operative treatment of pure allergic manifestations in nose and sinuses must be scrupulously avoided. Bearing in mind, however, the frequent coexistence of allergy and infection in the same individual (Mullin,[20] Hansel,[21] Valy Menken[24]), the treatment of the allergic condition must include the treatment of the infection, be that in the sinuses, teeth, tonsils or any other part of the body.

Knowing the rapid and extreme swelling of the allergic mucosa with its tendency to polyposis, great care will be exercised in nasal surgery, with this exception, that where allergy and sinus infection co-exist, the same rules as to the care and treatment of this sinus infection will obtain as in the nonallergic individual.

Before closing, mention might be made of the value of free iodin and calcium therapy (Feldman[25]) as an adjunct in the treatment of allergic states.

WELLS BUILDING.

BIBLIOGRAPHY.

1. Hilding, Anderson: The Physiology of Drainage of Nasal Mucus. I. The Flow of the Mucus Currents Through the Drainage System of the Nasal Mucosa and Its Relation to Ciliary Activity. Arch. of Otolaryng., 15:92:1932.

2. Hilding, Anderson: The Physiology of Drainage of Nasal Mucus: II. Cilia and Mucin in the Mechanical Defense of the Nasal Mucosa. ANNALS OF OTOLOGY, RHINOLOGY AND LARYNGOLOGY, 41:52:1932.

3. Proetz, Arthur W.: Some Intimate Studies of Nasal Function, Their Bearing on Diagnosis and Treatment. ANNALS OF OTOLOGY, RHINOLOGY AND LARYNGOLOGY, 41:125:1932.

4. Harkness, G. F.: Reactions to Infections. Transact. Am. Laryng., Rhinol. and Otol. Soc., p. 487, 1932.

5. Linton, C. S.: Infection and Resistance in Upper Respiratory Mucosa. Laryngoscope, April, 1933.

6. Schmidt, P.: Predisposition to Colds. J. A. M. A., February 4, 1933.

7. Lierle, D. M., and Moore, P. M.: Effects of Drugs on the Ciliary Activity of Mucosa of Upper Respiratory Tract. Arch. Oto-Laryng., Vol. 19, p. 55, 1934.

8. Proetz, Arthur W.: Displacement. Annals Publ. Co., St. Louis, 1931.

9. Frazee, J. R.: Diagnosis and Treatment of Sinuses by Displacement Method. Arch. Oto-Laryng., April, 1933.

10. Watson-Williams, Patrik: Chronic Nasal Sinusitis. Wm. Wood & Co., New York, 1930.

11. Van Alyea, O. E.: Irrigation in the Treatment of the Frontal Sinus. Arch. Oto-Laryng., Vol. 19, p. 224, 1934.

12. Shea, John J. Helping the Sinus Patient. Trans. Amer. L., R. and O. Soc., p. 413, 1932.

13. Jarvis, D. C.: The Upper Respiratory Tract as a Guide to Nutritional Disasters. Trans. Amer. L., R. and O. Soc., p. 275, 1930.

14. Cody, C. C.: The Relation of Vitamins A, D, B and G to Oto-Laryngology. Trans. Am. L., R. and O. Soc., p. 239, 1932.

15. Fenton, Ralph, and Larsell, Olof: Some Experimental and Clinical Observations on the "Reticulo-endothelial" Components of the Accessory Sinus Mucosa. Trans. Am. Acad. O. and O., p. 225, 1931.

16. Mithoefer, Wm.: The Use of Omnadin in Oto-Laryngology. ANNALS OF OTOLOGY, RHINOLOGY AND LARYNGOLOGY, Vol. 40, p. 26, 1931.

17. Besredka, A.: Are Antiviruses Specific? The Journ. of Immunology, Vol. 23, p. 349, 1932.

18. Kolmer, John A.: Bronchial Disinfection and Immunization. Med. Clin. North Amer., p. 617, November, 1933.

19. Mulso, John: Personal Communication.

20. Mullin, William: Allergy. Trans. Amer. L., R. and O. Soc., p. 384, 1932.

21. Hansel, F. K.: Allergy and Its Relation to Acute and Chronic Diseases of the Nose and Throat and Paranasal Sinuses in Children. Trans. Amer. L., R. and O. Soc., p. 511, 1932.

22. Rowe, Albert H.: Food Allergy. Lea and Febiger, Philadelphia, 1931.

23. Vaughn, Warren T.: The Diagnostic Program in Food Allergy. Amer. Journ. Med. Sciences, 182, p. 459, 1931.

24. Menken, Valy: Studies on Inflammation. IV. Fixation of Foreign Proteid at Site of Inflammation. Journ. Exp. Med., 52, pp. 201-213, 1930.

25. Feldman, Paul: Calciumtherapie in der Hals-Nasen-Ohrenheilkunde. Klin. Wochenschr., p. 475, March 25, 1933.

LXXXI.

CONSERVATIVE TREATMENT OF THE PHARYNX.*

JOHN W. CARMACK, M. D.,

INDIANAPOLIS.

Observing clinicians, since medical history is recorded, have considered the mouth and pharynx "the mirror of the body." Clinical medicine and research are constantly corroborating the observation that most pharyngeal pathology is secondary to disease in the nose and sinuses, the lower respiratory tract, or to some general systemic disturbance.

Pharyngeal diseases from a consideration of management and treatment may be classified as:

1. Primary infections implanted upon the pharyngeal mucosa by air or food borne organisms.

2. Pathology due to continued infection in the nose or lower respiratory tract or in the lymphoid or grandular structures in the pharynx itself.

3. Noninfectious irritants.

4. Pharyngeal manifestations of general alterations or diseases.

5. New growths.

When considering treatment, the nasopharynx, the oropharynx, and the hypopharynx must, to some extent, be considered as distinct entities, since they differ in anatomy and function.

The nasopharynx is essentially a part of the nose, being lined by the same ciliated, mucous covered membrane, which must always be respected. It contains structures of distinct importance, such as the pharyngeal end of the eustachian tube and the adenoid. Its function is to convey air, supplement the local defense mechanism of the nose proper, permit aeration and drainage of the ear cavities and, by its musculature, provide a barrier between the nose and pharynx proper. The wall of the nasopharynx being placed at almost right angles to

*From the Department of Otolaryngology, Indiana University School of Medicine.

Presented as part of Symposium on The Conservative Treatment of the Nose, Throat and Ear before the annual meeting of the American Academy of Ophthalmology and Otolaryngology, Chicago, September 13, 1934.

the nose, is subjected to maximum trauma from air currents and irritating substances in the inspired air. Because of natural architecture, nasal discharge is also directed into the nasopharynx, frequently with continued infection or irritation. The adenoid tissue, usually the first part of the pharyngeal lymphoid ring to become an active offender from chronic infection or hypertrophy, may continue infection or obstruction or, by adherence of its lobes, provide the necessary cavity for a Thronwald's abscess.

The above recital of architecture and responsibilities of the nasopharynx is made to emphasize the importance of an area which too often is grossly and radically treated by the application of toxic, irritating drugs or permanently damaged by careless removal of adenoid tissue.

The oropharynx, highly vascularized and enervated, is assigned the double duty of transmitting air and food. It is subjected to infections and trauma by way of the mouth, as well as from the nose and nasopharynx. It is lined by nonciliated squamous type epithelium, which permits of certain therapeutic liberties, not applicable to the nasopharynx. The oropharynx contains the faucial tonsils, which are usually the second part of the lymphoid ring to lose their usefulness in a breakdown of pharyngeal lymphoid defense. It also contains innumerable lymph nodes and glands, which may become hypertrophied or infected.

The hypopharynx assumes the highly specialized function of diverting food into the squamous cell lined esophagus, and air into the trachea, with its columnar ciliated epithelial lining. The structures of greatest importance in this region, from the standpoint of treatment, are the larynx and the tonsillar portion of the lymphoid ring.

Acute pharyngeal infections, resulting from an overpowering of the local and general defense by a virulent organism, whether it be a so-called cold infection, influenza, a milk-borne streptococcus or one of the acute exanthemata, is largely a matter of intensive general treatment. The extreme vascularity of the pharyngeal walls favor rapid absorption of toxic material, and the great number of mucous glands and lymphoid tissue in the pharynx, if involved, tend to increase and continue the toxemia. Complete rest with proper elimination and supportive treatment is of major importance. Local treatment, while of secondary importance, can assist in limiting the infection and add comfort to the patient. Local treatment is of value, both from the point of bacterial destruction and symptomatic relief.

The nasopharynx is usually the first portion involved in airborne infections, and is least amenable to local treatment in the acute stage, since the same anatomic defense mechanism must be conserved as in the nose. The application of toxic or irritating drugs to this area in acute infections, with their resulting reaction, depresses the local defense and may easily extend the infection into the eustachian tubes and middle ear.

Local treatment of acute infections in the oropharynx is more conducive to good results, with less likelihood of interfering with the local defense mechanism. It has in the past been the custom, however, to use drugs of a caustic nature as bactericides, such as strong silver solutions, iodin, etc. This practice cannot be supported either theoretically or in practice in the light of our present knowledge of tissue defense. The use of noncorrosive bactericides, if used frequently as hot gargles, is probably beneficial. These, however, must be used at frequent intervals to provide the bactericidal effect as continuously as possible, and doubtless the heat is as effective as the bactericide. The use of weak phenol solutions of noncoagulating dilution is antiseptic and adds much to the patient's comfort by their local anesthetic effect. As a local application, all the known antiseptics have been used, which in itself is an admission that none are entirely satisfactory. One of the most effective agents for application to an acutely inflamed oropharynx is a 5 to 10 per cent solution of guaiacol in glycerin. In addition to being an effective antiseptic, guaiacol produces a pleasing anesthetic effect in the throat for a considerable length of time, although its immediate effect is a distinct burning sensation. When applied to a mucous membrane, this mixture is retained for a prolonged period to continue its bactericidal effect.

Acute laryngeal infections also must be treated with due consideration of the existing anatomy. The tendency for edema to occur in the laryngeal mucosa, following acute pyogenic infections, make attempts at its prevention of first importance. This can best be done by absolute voice rest, the use of drugs for mild, general and tracheal sedation, the avoidance of irritating drugs in the larynx, and the use of massive cold applications over the larynx. Marked edema of the larynx from pyogenic infection is not infrequent in children, often to the point of dangerous asphyxia. When dangerous asphyxia is imminent, the logical procedure is low tracheotomy to prevent general exhaustion and fluid accumulation in the lung spaces. Intubation or bronchoscopy in these cases produces a high percentage of laryngeal strictures.

Chronic pharyngeal pathology, such as chronic infective pharyngitis, pharyngitis sicca and chronic laryngitis are purely symptoms, the various causes of which must be ascertained and eliminated if possible. Probably the most frequent cause is chronic nasal and sinus disease, with recurrent or continued infections in the numerous mucus glands and lymph follicles in the pharyngeal wall. Local treatment indicated in these conditions is mild stimulation of the mucus glands. This is best accomplished in the pharynx by silver nitrate in 2 to 5 per cent solutions. In chronic laryngitis, the instillation of 3 per cent creosote in olive oil is particularly effective. Occasionally the mucus glands and small lymph follicles of the pharynx become chronically infected, hypertrophy takes place, and these remain as continued sources of irritation after the nasal or other primary infection is removed. Surprisingly few of these remain, however, if all nasal or other factors are eliminated so that conservatism calls for repeated search for etiologic factors. If these cannot be found, destruction of the infected glands or lymph nodes are necessary by surgical removal, careful cauterization with chemicals or by electrocoagulation.

Pharyngeal pathology, due to infection in the lymphoid structures in the pharynx, a subject of major importance in this discussion, as it is concerned with the tonsil and adenoid problem. Acute infections in the tonsils and adenoid are essentially a condition requiring general treatment, with local attention, as described for an acute infectious pharyngitis. It is not a conservative or logical act to traumatize an acute or chronically infected mass of lymphoid tissue by attempts at curettage or suction of the tonsil crypts. If a distinct abscess is present, free incision is indicated for drainage.

X-ray irradiation of some chronic pharyngeal diseases has an important place in therapy. Its value in the treatment of infected lymphoid structures is to reduce the amount of lymphoid tissue and facilitate drainage from unclosed crypts. It is valuable as an aid in the occasional case where surgery may be contraindicated, but it does not entirely eliminate infection. Irradiation is particularly valuable in some chronic pharyngeal infections, such as carriers of diphtheria, Vincent's or actinomycosis.

Regarding the local or conservative attitude toward the removal of lymphoid tissue from the pharynx. In spite of the fact that the adenoid and tonsils are apparently not true lymph glands, there is evidence that when in a reasonably healthy condition they act as part of the first line of defense in the early years, when the individual is building a general defense against the common infections. There can be no logical argument in favor of tonsil and adenoid removal

except that they have not only lost their defensive power but actually harbor infection, which is being disseminated, or they are sufficiently hypertrophied to produce obstruction. Hypertrophy of the tonsil and adenoid may be either the result of infection or of a systemic glandular or chemical dyscrasia, which should be determined and corrected. When evidence accumulates that these structures have been overwhelmed, as complete a surgical removal as possible is the method of conservatism.

The adenoid is the earliest and most serious offender in younger children, and its removal, without permanent damage to the nasopharynx, is a difficult procedure. Conservation of the basal portion of the pharyngeal mucosa underlying the adenoid tissue is essential to prevent scar formation and an irritable nasopharynx in the future.

Conservative treatment of chronically diseased tonsils calls for complete surgical extirpation, as against electrosurgery, because of its higher percentage of complete removal and satisfactory results.

Specific infectious diseases, common to the pharynx and larynx, are tuberculosis, syphilis, Vincent's infections and, in certain sections, actinomycosis.

Tuberculous infections in the pharyngeal wall or in the larynx are best treated locally by electrosurgery, either by removal of granuloma or surface cautery on the walls of the pharynx or multiple stab puncture in the larynx. The varied therapeutic agents, such as chaulmoogra oil, acids, heliotherapy, etc., used locally in these infections, have not proved of sufficient value to consider their use as conservative.

Syphilitic lesions of the larynx are only local manifestations of a general disease and must be treated in a general way.

Vincent's infections in the pharynx can, in the majority of instances, be eliminated by frequent application of sodium perborate to the infected area. If, however, the infection is severe or inaccessible, one should resort at once to moderate doses of arsphenamin intravenously. The local application of this drug is of exceedingly doubtful value.

Noninfectious irritants of any nature, the most common being tobacco smoke, are a source of irritation to the pharynx in certain individuals. This can be demonstrated by observing an irritable pharynx with a cough subside after smoking is eliminated. Elimination of smoke as an irritant is a valuable but little used therapeutic procedure in pharyngeal diseases. The addition of cooling drugs to cigar-

ettes, in the light of recent investigations of the effect of menthol on the respiratory mucosa, indicates further irritation from these.

We must not overlook the fact that many of the chronic pharynges, especially of the irritable conjestive type, often with great quantities of mucus secretion, is merely a superficial reaction of an altered general chemistry or the result of vascular disease, where circulatory power is impaired. The much discussed mucous membrane changes in the nose, due to altered general processes, have an older counterpart in the so-called rheumatic or gouty throat of the past and is deserving of constant thought in attempting to diagnose and treat pharyngeal diseases.

Recent developments in the treatment of malignancies in the pharynx, based upon the pathologic picture present, have added much to conservatism and offer greater possibilities of a cure. Every pharyngeal growth observed demands immediate biopsy, as early diagnosis is the essence of conservatism. Irradiation of a malignancy, for limitation before biopsy or any surgical procedure, would be ideal. This is not practical as a routine, but should be practiced in all highly suspicious cases. The present day classifications of carcinoma cannot be accepted as a positive indication of radiosensitivity but, generally speaking, the epithelial growths classified as type one and two cell construction are not radiosensitive, and conservatively demand wide surgical removal with irradiation following for its limited effect. Carcinomas classified as type three and four are apt to be more radiosensitive. To this latter group also may be added sarcoma and lymphoepithelioma. These tumors are best treated by prolonged fractional irradiation to a saturation point, combining both X-ray and radium. If, however, massive growths are present, their removal facilitates irradiation. Conservative management of laryngeal malignancies calls for the same type of prolonged fractional irradiation as the pharyngeal malignancies, but is demanding more than ever early open operation for complete surgical removal rather than by endoscopy.

505 HUME-MANSUR BLDG.

LXXXII.

CONSERVATIVE TREATMENT IN DISEASES OF THE EAR.*

JAMES A. BABBITT, M. D.,

PHILADELPHIA.

Medical and surgical treatment in otolaryngology would seem to have been in a peculiarly transitional state for the last decade. Several important factors have contributed to this.

First, the growing realization that the influence of nutrition and biochemistry upon all upper respiratory pathology has not only been underrated but too often ignored. There is sufficient evidence to believe that research investigation in this field, already well advanced, may change the entire perspective of both routine and surgical measures in the not too distant future, and that adequately regulated body chemistry will supplant much that is being imperfectly done at the treatment table—the time worn analogy of reaching the trouble at its source.

Second, the almost finality which has now been attained in the technic of radical surgery. Perhaps sufficient time has now elapsed to balance the ledger and estimate experience in end results. This has automatically occurred in large degree, as one could almost forecast the popular trend in radical mastoid and sinus surgery.

Third, the better understanding of allergic reaction and its intimate relation to medical and surgical decision. Probably only the threshold of this important correlation has yet been passed, and its breadth of application is not yet sure. The intranasal and sinus birthright to allergic manifestation should claim priority in this symposium discussion.

Fourth, the confusing advent of manifold newer antiseptic preparations and the rapid supplanting of time honored therapeusis now experimentally proved inefficient. It would seem that medical as well as surgical treatment in recent years has reached a radical stage.

The honest specialist is confronted with such queries as this. Are routine nasal irrigations, oropharyngeal applications, tubal treat-

*Presented as part of Symposium on Conservative Treatment of the Nose, Throat and Ear, before the American Academy of Ophthalmology and Otolaryngology, September, 1934.

ment and massage overdone? This and other symposiums on the conservative handling of the ear, nose and throat are a response to the transitional unrest cited above and should further stimulate biochemic research.

It must not be interpreted from the foregoing that the intention of this paper is to champion the neurologic handling of ear pathology. One must be in perfect agreement with Shambaugh,[1] who summed up his discussion of clinical problems in otitis media by saying that no form of local treatment can ever be expected to cure a running ear where an indication exists for radical surgical measures.

It is the purpose here to discuss borderline cases and argue where possible for the conservative plan. The symposium title was made "conservative" rather than "nonsurgical" to broaden this discussion. No effort will be made to review the voluminous literature on the subject, which all may read. This will be simply a chronicle of clinical observation and experience.

The three most important conditions in the ear to be considered from the conservative standpoint are infection of the external canal, acute and chronic middle ear involvement, and mastoid infection. Labyrinthine infection, tubal stenosis and progressive deafness might indeed be added, for there is wide variation in routine, but the latter conditions are not so germane to the title.

The greatest safeguard for conservatism rests in the preliminary history and record of each patient. Nothing but emergency surgery, and in the case of the ear seldom that, should prevent a careful analysis of the patient's own and family history, habits of life, response to variations in climate, dietary regime, elimination, associated medical conditions, depressive influences and idiosyncrasies. To this should be added minute laboratory study, blood and urine studies, including the daily von Schilling shift of immature neutrophiles, and basal metabolism. Ear tests, tuning fork and audiometer, should be taken when feasible, regardless of known deafness, and a preliminary caloric test for vestibular tract may give you surprising leads. Voluntary nystagmus, gross neurologic reactions and the nasopharyngoscopic picture of the pharyngeal opening of the eustachian tube should be noted. In even doubtful cases mastoid roentgenograms, eye grounds and fields, and a cautionary blood culture should be taken. Allergic reactions, even in a study of the ears, are appropriate. One cannot visit the offices of some of our finest otologists without being impressed with their care and precision in collection of almost voluminous detail in their masterly methods of analysis.

This careful preliminary study in even simple ear conditions is important for the following good reasons: First, valuable data are on record for comparison in later ear study and treatment; second, intracranial mischief is often discovered from routine study, even in the semiquiescent stage; third, information is often derived which shows that this patient does not really belong in otologic hands and the proper reference can be made; fourth, the ear with its tinnitus, vertigo and hallucination hearing, very frequently has a psychic bearing. The patient's confidence sequent to the realization of a cautious study is well worth having, for optimism is therapeutic in itself. One grave caution is the question of finance. Laboratory work does cost money, but the otologist should have sufficient influence to obtain this for patients of limited means. Fifth, otolaryngologists wish to share their achievements for the benefit of science. Results of study and experience are worth but little unless fortified by accurate and trustworthy records. Sixth, as a final reason for this somewhat lengthy preamble to conservative treatment, specialists in every field may be roughly divided into two groups—one with radical, one with conservative bent. If one has the operative habit, his subconscious reaction is operative. This is at least effective but not always protective to the patient. If he is at heart conservative or wishes to become conservative, he will emphasize this preliminary investigation and be less liable to interpret basic conditions by superficial irregularities.

INFECTIONS IN THE EXTERNAL CANAL.

Three conditions might be pictured in discussion here: (a) Furunculosis of the canal; (b) disorders of the epithelium; (c) disturbances arising from the presence of exostoses or other malformations.

(a) *Furunculosis of the Canal.*—This is too often not a simple matter and tries both physician and patient. The long and deep incision to the periosteum in the early stages of this disorder is usually neither wise nor conservative. Here as elsewhere nature needs a period of time to wall off and protect surrounding tissues. While direct transmission to middle ear, mastoid or meninges is perhaps unlikely, there is no evidence to prove that surgery will greatly shorten the old time antiseptic or poulticing methods, persisted in to absolute pointing and well nigh automatic elimination. This is not in the same category as the infection of the upper lip and angle of the nasal alæ, but conservatism invites culture, vaccine and systemic treatment to build up furuncular resistance.

(b) *Disorders of the Epithelium.*—There is some consensus of feeling that infections involving the lining membranes of the ear canal should be put under dermatologic supervision, as the lesions can be so manifold. It should always be borne in mind how sensitive is the external ear canal—how easily vascular blebs are formed in topical manipulation. It would be conservative to avoid unnecessary paracentesis of the tympanic membrane because of failure to diagnose myringitis bullosa and herpes of the drumhead, canal mycosis or rupture of abscess through fissure of Santorini, as pointed out by Hunter.[2]

(c) *Exostoses and Other Malformations.*—Exostosis deep in the canal may cause much concern and the surgical temptation is great. Fortunate is the surgeon who makes approach through postauricular route rather than through the canal if he must operate. Usually conditions present are complicated by the abnormal anatomy rather than directly caused by it—careful diagnosis and treatment will more often obviate the necessity of interference.

ACUTE OTITIS MEDIA.

The two most popular subjects in any otolaryngologic session are removal of the faucial tonsils and incision (paracentesis) of the tympanic membrane. The tympanic problem revolves about the question as to when the membrane should be opened. There are many variations in acute intratympanic involvement, from the simple injection, varying degrees of myringitis, serous fluid level, with or without air bubbles, to the massive bulging with toxemia and febrile reaction where there is no debate.

With the evidence of definite mastoiditis, delay in abortive measures would be unjustifiable. Many cases of mastoiditis doubtless occur from ultraconservative delay, and some doubtless occur without obvious middle ear disturbance. Nevertheless, countless middle ears are opened unnecessarily, often resulting in chronic suppurative otitis media. Many argue that a middle ear with catarrhal serous fluid, and not drained, contributes to middle ear deafness. Careful study of many ears in our office with tuning fork and audiogram check-ups would, however, tend to disprove this.

In doubtful ears, with neither mastoid nor systemic threat, disregard the simple hyperemia and treat as a myringitis. Packard once pointed out in a ward review that the short process and malleus line in clear relief tends to negate posttympanic fluid. Why not immediately attack the pharyngeal end of the tube—ephedrin, cocain or

other measures to obtain tubal drainage, and await results? Even if tympanic opening be necessary, repeated incisions are of doubtful value as the middle ear becomes filled with scleroses and granulations.

Chronic Suppurative Otitis Media.—It is impossible to avoid linking this with the question of the radical mastoid operation, so often employed for its relief. The study of chronic suppurative involvement of the middle ear requires some review of nature's basic efforts in relief, and one becomes intimately involved in discussion of Aschoff's and Wittmaack's theories of infantile otitis media and sequence of the diploetic and sclerotic mastoid states, which in principle prevent the occurrence of a coalescent mastoid involvement, which has been so admirably presented by Almour.[3] In infantile otitis media neonatorum, due to early respiratory forcing of meconium and other substances into the middle ear, the middle ear, antrum and mastoid become filled with hyperplastic connective tissue, and pneumatization is halted with a resultant sclerotic mastoid; in the true infantile suppurative otitis media, due to bacterial invasion from the eustachian tube or through blood or lymph from an infected external canal, fibrosis occurs with marked contraction of the subepithelial embryonal connective tissue, and again a halt of pneumatization occurs with a resultant diploetic mastoid. Both of these conditions, with various interesting defense mechanisms in the different types of tympanic perforation, change the middle ear suppuration to a somewhat local problem and naturally invite the multitudinous methods and medicaments presented for its control. From a conservative standpoint this situation places the radical mastoid in a position of last resort. Chronic suppurative otitis media may be chronic, recurrent or intermittent, but this does not greatly change the principles of treatment.

From the conservative position and assuming that in every middle ear invasion there is at least initial mastoid involvement, antrum or anterior lateral cells, the first thing to do is to determine by accurate x-ray and other diagnostic measures the sclerotic, diploetic or other states of the mastoid. The second point is to determine the defense mechanism in tympanic membrane. A central large type perforation should tend toward self-limitation when proliferating mucosal membrane comes into conjunction with marginal squamous celled activity in the perforation wall. This type, according to the work of Asherson,[4] alone responds favorably to the zinc ionization treatment which might shorten nature's more tedious repair.

The third step in conservative procedure would be the effort to obtain nonsurgically the results of the radical method, which would

customarily be either ossiculectomy, the radical or modified radical mastoidectomy, intratympanic mastoid antrum approach as in the operation of Tobey,[5] and perhaps, by your permission, some such modified attic drainage as that advocated by the writer.[6] This effort should be twofold, to produce drainage and apply antisepsis. In suppurative involvement of the middle ear, the cavity has become filled with adhesions, scleroses, organized granulations and fibroses until the initial drainage has become a narrow, tortuous and irregular canal. Remembering that destruction of tissue and retained fluid will tend to increase the discharge, an effort should be made to clear away cholesteatoma, crusts and other débris, by peroxid, alcoholic, boric and glycerinated solutions, and then make sure that the drainage exit is clear. Dilating the existing exit in safe areas such as the posterior quadrant can do no harm. New membranes will form rapidly once the infective element is removed. A cardinal point in the use of antiseptic agents—no amount of antiseptic treatment per the auricular canal route will be of any avail unless it actually reaches the intratympanic infection, and therein lies the value of actual irrigation by attic cannula. Iodin powder or other agent must pass through the tympanic opening to produce result. X-ray radiation, ultraviolet and infrared rays, high frequency current, mercury quartz light and similar methods may prove the exception to this rule, but their results have not yet been sufficiently attested in experienced hands to fully prove their efficacy. Many antiseptic preparations have been used, of which the most popular has been the iodin powder advocated by Lederman.[7] Others have prepared similar powders in their own laboratories. Hurd[8] suggests surgical diathermy to remove adhesions and infected lymphoid follicles around the mouth of the eustachian tube. Nelson,[9] ephedrin solution directly in middle ear; Hetrick,[10] postural drainage; Fox,[11] dental drying rolls; Kirkland,[12] chromic acid; Watson-Williams,[13] magnesium sulphate crystals; Shaheen[14] and others, subcutaneous proteid injections. Disulpharmin,[15] picratal,[16] oxyquinalin tartrate,[17] meroxyl and acetic acid[18] have all been the subject of favorable experience reports. All these procedures carry but one suggestion—cases are individual. Treatment, both systemic and local, will depend for success largely upon the patience and skill of the otologist rather than by any particular medication.

Mastoid Infection.—In mastoiditis, immediate operation is seldom required. There is usually sufficient time to study the patient and fortify his resistance. Many a mastoid is aborted by prompt and adequate care of the middle ear, and there is no evidence to indicate that when the patient has escaped operation by such treatment the

ultimate function of the ear has been injured. An acute mastoid in experienced hands is given an opportunity for walling off during a period which might vary from one or two days to even weeks. The danger of rapid intracranial invasion from too hasty operation in the presence of subacute meningeal symptoms of toxic character will thus be avoided. Such a delay is trying for the surgeon, patient and his friends and often induces a change of otologist. Experience has demonstrated that ultimate confidence is gained from a firm stand.

In this transitional period, with the prospect of great awakening from scientific research, now well in hand, it behooves clever otolaryngologists to stand in the front rank, grasp proven guide posts and at least present the conservative rather than the surgical trend of mind.

1912 SPRUCE STREET.

BIBLIOGRAPHY.

1. Shambaugh, G. E.: The Clinical Problems of Chronic Suppurative Otitis Media. Trans. Amer. Otol. Soc., 20:132-139, 1930.

2. Hunter, R. J.: Otitis Externa. The Penna. Med. J., March, 1934, pp. 477-480.

3. Almour, Ralph: The Significance of the Squamous Epithelium in the Cause and Repair of Chronic Middle Ear Disease. Trans. Amer. Acad. Ophth. and Otolaryn., pp. 357-377, 1930.

4. Asherson, N.: Zinc Ionization in the Treatment of Chronic Suppurative Otitis Media. Laryngoscope, 42:141-147, Feb., 1932.

5. Tobey, Geo. L., Jr.: Chronic Suppurative Otitis Media. Trans. Amer. Otol. Soc., Vol. 20, pp. 140-149, May, 1930.

6. Babbitt, J. A.: A Modified Attic Drainage in Chronic Suppurative Otitis Media: Preliminary Report. ANNALS OF OTOLOGY, RHINOLOGY AND LARYNGOLOGY, 40:348-359 (June), 1931.

7. Lederman, M. D.: Nonsurgical Dry Treatment of Chronic Suppurative Otitis Media with Iodin Powder (Sulzberger). Trans. Amer. Laryn., Rhin. and Otol., 36:44-63, 1930.

8. Hurd, L. M.: The Use of Surgical Diathermy About the Mouth of the Eustachian Tube for Infection and Catarrh of the Middle Ear. Trans. A., L., R. and O., 37:331-334, 1931.

9. Nelson, R. F.: Ephedrin Sulphate: Its Use in Otitis Media. Arch. Otolaryn., 15:444, March, 1932.

10. Hetrick, J. A. W.: The Use of Posture in the Treatment of Acute Otitis Media. J. Ophthal., Otol. and Laryn., 31:183-188, June, 1927.

11. Fox, M. C.: Cotton Dental Rolls for Ear Drains. Northwest Med. J., 28: 173-176, 1929.

12. Kirkland, T. S.: The Use of Chromic Acid in Aural Suppuration. Med. J. Australia, 1,684, May, 1930.

13. Watson-Williams, E.: Otorrhea. Brit. Med. J., 2:47-50, July, 1933.

14. Shaheen, H. B.: A Note on the Treatment of Chronic Suppuration of the Middle Ear by the Subcutaneous or Intramusclar Injection of Boiled Milk. J. of Laryn. and Otol., 44:27, 1929.

15. Callison, James F.: Chronic Purulent Otitis Media: Use of a New Antiseptic with Satisfactory Results. Med. J. and Rec., 1:529, May, 1930.

16. Vanstane, J. R.: Some Uses of Picratol with Special Reference to Chronic Otitis Media. Illinois Med. J., 63:168-172, Feb., 1933.

17. Stein, Simon: A New Organic Compound for Use in Chronic Ear Suppuration. Laryngoscope, 42:219-224, March, 1932.

18. Schwartz, E. M.: Chemotherapy in Otitis Media. ANNALS OF OTOLOGY, RHINOLOGY AND LARYNGOLOGY, 34:932-937, Sept., 1925.

LXXXIII.

ABSCESS OF THE LARYNX AND ITS TREATMENT.*

JOHN DEVEREUX KERNAN, M. D.,

HENRY P. SCHUGT, M. D.,

NEW YORK.

Laryngeal abscess is an uncommon and dangerous disease. It used to occur more frequently in former years as a complication of typhoid fever. More recently, however, it has become less frequent. The physician treating such cases is often confronted by a complicated problem. A careful study of all previously published cases is the best way of learning how such cases can be diagnosed and treated. We, therefore, believe that all cases should be reported in the literature. O. Mayer,[1] who published a comprehensive paper in 1931 on abscess of the larynx, found only thirty such cases occurring in a period of eighteen years among a total of 73,413 patients examined.

We have already described two cases[2] fitting into this category, in a previous publication which dealt chiefly with those rare forms of laryngeal abscesses in which the pyriform fossa is involved. Since then we have succeeded in gathering a total of eight cases which we consider suitable for demonstrating the entire field of laryngeal abscess in more detail, and from which we feel that we have gained a certain amount of experience in diagnosing and treating this condition. We will endeavor to show in this paper how other workers have previously handled these cases, and also describe the methods which we have used in our cases and which differ in part from previously described methods. We have chosen the term laryngeal abscess because it is the most frequent form in which purulent affections of the larynx occur. Formerly, various terms were used to describe this condition and the following ones were used indiscriminately: Edema laryngis, edema glottidis, laryngitis submucosa, laryngitis edematosa, laryngitis phlegmonosa, angina laryngea infiltrata, laryngitis seropurulenta, angina laryngea edematosa and abscessus laryngis. Kuttner[3] was the first to demonstrate that the process currently termed laryngeal abscess, as well as the majority of the processes designated by the foregoing terms, arise from a so-called laryngitis submucosa,

*Read before the annual meeting of the American Laryngological Association, Cleveland, June 9th, 1934.

and that therefore a majority of the above mentioned terms are confusing and superfluous. Today the following terms remain in common use: (1) Edema of the larynx, (2) perichondritis of the larynx, (3) abscess of the larynx and (4) phlegmon of the larynx. These four conditions are, however, more or less the same disease in various stages of development.

ETIOLOGY.

The traumatic form of this disease is the only one that has its origin in the larynx itself. In studying the histories of a series of cases one finds that in only a few instances did the illness begin in conjunction with an injury resulting from swallowing a foreign body. Rarely can the presence of such a foreign body be actually demonstrated. Seifert[4] described a case were a fishbone was found in the discharging pus from an incised perilaryngeal phlegmon. Other cases have resulted from external trauma (blows or lacerations) to the larynx. Finally, the presence of tumors in the larynx (carcinomata, for example) can cause abscess formation by breaking down and becoming infected. All other cases of abscess of the larynx must be considered as secondary to other infections, either of a general nature or else of infections in the vicinity of the larynx itself. Typhoid was formerly observed to be the most common infection. In this disease the laryngeal complication occurs most often during the period of convalescence. It is still debatable whether or not the involvement of the larynx takes place via the blood stream, as many claim. Some authors assume that a sort of decubital ulcer develops as a result of the prolonged flat position of the body, which causes the larynx to press back onto the pharyngeal wall, and leads to infection of the laryngeal structure. Keen[5] was the first, in 1877 and 1898, to publish comprehensive papers on the laryngeal complications of typhoid. Besides the articles of Keen, the more important papers in this field have been published by Chevalier Jackson,[6] Rieser[7] and Hinsberg.[8] In addition to typhoid, scarlet fever and measles (Zarfl[9]), typhus, septicemia (Imhofer[10]), puerperal sepsis, erysipelas (Zarfl[9]), gonorrhea (Birkett[11]) and syphilis must be considered as possible infections having such laryngeal complications. In infancy, an infection of the umbilical blood vessels can occasionally cause a perichondritis of the larynx secondarily as a metastatic infection (Zarfl[9]). Forschner[12] described a case of phlegmon of the larynx which followed a middle ear infection.

More recently, the majority of the cases reported have followed either a grippe or occurred in connection with an acute tonsillitis and pharyngitis. In these primary, localized infections of the throat, the

infection can spread superficially and thus reach the larynx by direct continuity, or else the larynx may also be involved by way of the hematogenous route. The least likely route would be along the lymphatics, since those draining the tonsils are not directly connected with those draining the peri- and paralaryngeal tissues. On the contrary, the lymph channels from the tonsils and those from the larynx empty into the lymph nodes along the large vessels of the neck, and a retrograde infection could hardly be expected to take place. MacMahon,[13] however, on the basis of his histologic studies, believes that he has proved that there is a stasis of the laryngeal lymphatics in acute tonsillitis, and that therefore a retrograde lymphangitis leading to phlegmon formation in the larynx can occur. Wiehe[14] assumes that a phlegmon of the larynx can occur in certain cases as a result of infection of tonsillar tissue present in the pyriform fossa. This tonsillar tissue is a finding not always present, and under normal conditions is so small that it can hardly be discerned with the naked eye. In the cases treated by us, and which we will describe more fully later on, we were able to demonstrate that one case (No. 2) was probably of traumatic origin, while the others developed from infection of the tissues of the vicinity of the larynx. In summing up, we can therefore say that abscesses and phlegmons of the larynx almost always occur secondarily to an infection elsewhere, with the exception of those cases of traumatic origin already mentioned in which the disease must be considered as having arisen in the larynx.

SYMPTOMS, DIAGNOSIS AND PATHOLOGY.

The symptoms and local findings in laryngeal abscess and similar infections have been well known since the classical description of Türk[15] and other later authors (Von Schroetter[16]), Hajek[17] and Hinsberg.[8] All of these descriptions have dealt chiefly with those affections of the larynx occurring with typhoid, whereas in our cases, and also in most of the cases occurring in recent years, grippe and local throat infections have been the underlying cause, with the involvement of the larynx secondary to them.

Pain is the first symptom to appear and may be spontaneous or else appear only on pressure or on swallowing. It seems to be further down than the pain in tonsillitis and is associated wtih the sensation felt when a foreign body is present. This sensation of a foreign body is particularly evident when there is swelling of the lamina cricoidea. In that condition, typical pain can also be elicited by encircling the cricoid from behind. Hoarseness is seldom absent, but in some cases may not be very marked, especially if the cartilage is involved only on the outer side or if the abscess does not particularly involve the

inner structure of the larynx. This latter condition is found, for example, in submucous abscesses of the larynx in which the pus opens into the pyriform fossa, as we shall demonstrate in our cases.

Dyspnea is often present and is one of the most dangerous symptoms. One seldom finds the epiglottis involved alone, but if it is, these cases are generally favorable. (Case No. 1.) There is usually a large edematous swelling to be seen, which subsides rapidly on evacuating the pus. Severe involvement of the epiglottis with necrosis occurs but rarely in the purulent form, as the cartilage of the epiglottis is very strong and resistant.

When the arytenoids are involved, the disease manifests itself (1) through swelling and (2) through the limitation of motion. Inasmuch as the laryngeal cartilages in the adult are all ossified to a large degree, a real osteomyelitis can occur in the arytenoids as well as in any of the other laryngeal cartilages (O. Mayer[1]). If there is abscess formation, spontaneous evacuation of the abscess can take place either laterally into the pyriform fossa or anteriorly into the larynx. With a probe one can feel the denuded cartilage at the point of rupture of the abscess. These cases generally heal very rapidly. Such cases have been described in the literature by Chiari,[18] Schroetter,[16] Imhofer,[10] Von Heyman,[19] Levinstein,[20] Kuttner,[3] Jackson,[21] Hecht,[22] Tobold,[23] Verweyen[24] and Cruveilhier.[25]

If an osteomyelitis is present, the swelling often remains for quite a long time. A moderate swelling of the arytenoids, in which there is often only a submucous inflammation or a simple perichondritis, can subside without necrosis or abscess formation. There then sometimes remains a thickening and a fixation of the arytenoid cartilages. In other cases the cartilages or parts thereof are sloughed out as sequestra. The diagnosis is at times difficult when swelling is no longer present and only a limitation of movement of the arytenoid cartilage, with fixation in the midline, remains. The picture is then similar to a paralysis of the crico-arytenoidei postici (abductor paralysis) and is differentiated from that condition by the degree of midline fixation. In the perichondritis of the arytenoid the fixation is absolute, whereas in the abductor paralysis adduction is still present.

In children and young adults up to the age of twenty, involvement of the thyroid cartilage usually appears as a submucous laryngitis which then goes on to a perichondritis and possibly abscess formation. In adults over the age of twenty, the condition is usually a periostitis or an osteomyelitis, as the thyroid cartilage is ossified to a large extent (O. Mayer[1]). The ossification consists of two thin bony layers on the outer and inner surface, together with a loose

layer of cancellous bone rich in marrow. It is easy to diagnose a perichondritis of the thyroid cartilage, if there is a definite, tender swelling externally over the cartilage. Cases have been described in which the infection, starting on the inner surface, had broken through the cartilage and appeared externally. These cases have also occurred in small children (Zarfl,[9] Descortes[26] and McIntosh.[27]) It is in these laryngeal abscesses pointing to the outside, that a differential diagnosis must be made. The condition might be an abscess of the thyroid gland (Brueggemann[28]) or possibly an abscess of the prelaryngeal lymph node. This node lies anterior to the cricothyroid ligament. A prelaryngeal abscess of this type occasionally develops following intralaryngeal operations, particularly after operations of the false vocal cords. The infection spreads along the intralaryngeal lymphatics. Bekarian[29] described eighteen such cases and maintains that the majority of them are cold abscesses of tuberculous origin.

We have observed only one case of abscess of the prelaryngeal node, in which instance the abscess formed following cauterization of a tuberculous larynx (Kernan and Schugt[2]).

And finally in the differential diagnosis one must also think of an infected cyst of the thyroglossal duct.

Definite changes in the inside of the larynx are almost always present in abscesses of the thyroid cartilage, whether the abscess is in the acute or in the chronic stage. Swellings of the vocal cords and of the ventricular bands, subglottic swellings and occasional fistulæ near the commissure, occur. Sometimes the arytenoid cartilages are involved simultaneously with the thyroid cartilage, so that at first that symptom complex seems to dominate the picture. After the swelling of the arytenoids has subsided, the patients' complaints, consisting of slight external swelling and tenderness, are often so slight that it is difficult to persuade them to submit to an extensive external operation. In that event, the abscess of the thyroid cartilage can burrow downwards without showing many signs, and lead to a mediastinitis.

The pyriform fossa plays an important part in the diagnosis of abscess of the thyroid cartilage, but unfortunately little importance has been attached to it. We will describe several cases at the end of this paper in which the presence of an abscess could be surmised only through the appearance of the pyriform fossa.

Pieniazek[30] is the first and, in fact, the only one to mention the importance of the pyriform fossa in laryngeal abscesses, as far as we can determine from the literature. In infections of the thyroid

lamina the outer wall and floor of the pyriform fossa bulges upward because the upper and posterior portions of the thyroid cartilage form an unyielding wall externally (Fig. 17). A definite bulging of the outer wall of the pyriform fossa is pathognomonic of an exudative process on the lamina of the thyroid cartilage (Figs. 5, 11, 16, 17). If only the floor of the pyriform fossa bulges upwards, the underlying exudate may be present both in the vicinity of the thyroid cartilage as well as of the cricoid. In the latter case the arytenoid cartilage is immovable and its mucous membrane is definitely swollen.

In a true perichondritis of the inner portion of the thyroid cartilage, either one or both laminæ of the cartilage can be involved at the same time. If in addition to the laminæ the median portion of the thyroid cartilage is involved, a definite subglottic swelling over the anterior commissure of the larynx is present (Fig. 7), in addition to other laryngeal changes. These changes are swelling of the pharyngo-epiglottic ligaments and of the ventricular bands; the mobility of the arytenoids may be partially or entirely checked by the accumulation of exudate in the pyriform fossa. The vocal cords are usually obliterated by the swollen ventricular bands. Marked swelling of the ventricular bands indicates involvement of the thyroid cartilage, whereas swelling of the posterior wall of the larynx beneath the laryngeal aperture speaks more for involvement of the cricoid cartilage.

The diagnosis is much more certain if the infection is present further anteriorly—that is, near the junction of the two laminæ of the thyroid cartilage. Then one may find a swelling of both ventricular bands which may be so swollen as to touch each other anteriorly for a part of their length. Occasionally a swelling of the lingual surface of the epiglottis as well as of the vallecula is also present (Case No. 1). A prolapse of the ventricle may occur in long standing infections (Case No. 3, Figs. 7 and 9).

O. Mayer[1] has shown cases, examined histologically, in which a widespread periostitis has occurred together with a superficial perichondritis. In these infections the cartilage is involved in an entirely different manner than is the bone. The infection spreads either above or beneath the perichondrium in the cartilaginous portions, and the cartilage itself is involved only superficially. The abscess forms on the surface of the cartilage and only then, when the cartilage is completely surrounded by pus, does necrosis and sequestration take place. The reason for this is that the cartilage is deficient in blood vessels. In the bony portions of the cartilage the infection spreads more rapidly in the cancellous bone than in the periosteum. There

are also cases in which the abscess is limited to the submucosa but in which the infection may be fairly extensive. We were able to demonstrate, in cases 7 and 8, that such an abscess may be present for considerable time without involving the deeper layers (perichondrium and cartilage). In these cases the inner perichondrium was found to be intact on opening the thyroid cartilage (Fig. 18), but on incising this perichondrium there was a large abscess between it and the submucosa of the inner surface of the thyroid cartilage.

Albrecht[31] has described a case in a child, in which there was only a smooth swelling of the right ventricular band to be seen. Pressure on the right lamina of the thyroid cartilage was painful. The lamina of the thyroid cartilage was penetrated through an external incision and an encapsulated abscess filled with granulations found.

The cricoid cartilage is least often affected in abscess formation and perichondritis, but those cases in which it is affected have the most serious complications and are the most difficult to treat. One may find this characteristic picture: There is a marked swelling of the lamina cricoidea, and particularly of its pharyngeal surface, which bulges in a tumor-like swelling, pushing the entire larynx forward and thereby blocking the pharynx. Difficulty in swallowing manifests itself more completely than in any other form of inflammatory disease of the laryngeal cartilages. In addition, there is usually a swelling and fixation of the arytenoids if the infection involves the entire lamina cricoidea. If the infection is limited to one side of the lamina cricoidea, the opposite arytenoid is more or less freely movable. This occurs fairly often, because the lamina consists of two lateral bony nuclei separated by a cartilaginous central portion. (See Fig. 1.)

In order to determine the exact site of the infection, it is important to know how ossification takes place in the cricoid cartilage. This applies particularly to the osteomyelitic infections, which are found chiefly near these bony nuclei. Chievitz[32] was the first to accurately describe the ossification of the laryngeal cartilages, and for a description of this process in the cartilages other than the cricoid we refer you to the "Handbuch von Denker-Kahler, Vol. 1, page 379." In the cricoid cartilage, a bony nucleus first appears in the vicinity of the cricoarytenoid articulation, and is followed later by another bony nucleus in the cricothyroid articulation (Fig. 1). The base of the lamina cricoidea remains cartilaginous for a long time. It is this part which later forms the arch. It ossifies a great deal later on, whereas the ossification of the lamina has already become almost complete around the twentieth year. The cartilage is often much less affected by the inflammatory process than is the bone. The bone formed in

the lamina cricoidea consists of very fine cancellous tissue containing either red or fatty marrow, just as in the other bones throughout the body. There is often a thick cortical layer surrounding the cancellous bone.

It therefore follows that in adults the process in the cricoid is an osteomyelitis. An unilateral infection is most common, because, as previously mentioned, the bony nuclei are separated from each other. Since the infective area lies just beneath the cricoarytenoid articulation a fixation of the arytenoid is a common sequel. After the acute stage, which lasts about a week and manifests itself by swelling of the cricoid and a surrounding edema has quieted down, the inflammatory process can either subside and disappear or else continue further in the depths of the marrow cavity. It can even spread to the other arytenoid not previously involved, and the sudden dyspnea seen in some cases can certainly be attributed to such a spread. If the infection does involve the other arytenoid, the following picture is seen: There is a moderate swelling of the arytenoids and of the pharyngeal surface of the cricoid, together with swelling of the ventricular bands and apposition of the vocal cords in the midline. There is also a subglottic swelling along side of a swelling of the posterior wall of the larynx, but usually this cannot be seen, due to the apposition of the vocal cords in the midline.

There are cases in which an air passage sufficient to prevent asphyxiation remains, despite a complete sequestration and sloughing out of the lamina cricoidea. In these cases the infection may heal spontaneously with a sloughing off of necrotic bony and cartilaginous fragments and a formation of new bone to act as a prop for the remaining tissues. Every sign of infection may be absent in these cases and the diagnosis of a neurogenic paralysis of certain of the laryngeal muscles may be incorrectly made.

The laryngeal involvement usually appears to the examiner to be very much less in extent than it really is.

As already mentioned, it is especially in infections of the cricoid that dyspnea and asphyxiation occur unless an early tracheotomy is performed. Sudden death can occur in other forms of laryngeal abscesses, however, and need not be due to asphyxiation. Seifert[4] described five cases, of which four died suddenly without showing any signs of asphyxiation. He mentions two possibilities as the cause of the death in these cases. The first is cardiac failure of toxic origin and the other is a sudden paralysis of the respiratory center due to an excessive accumulation of carbon dioxid, without any accompanying signs of strangulation.

THERAPY.

Unfortunately there are many differences of opinion as to the treatment of perichondritis or abscess of the larynx. On the one hand are those authors advocating conservative treatment; on the other, those advocating the most radical surgical procedures. Since most of the cases occurring formerly resulted as complications of typhoid, and since in this infection several of the laryngeal cartilages were usually affected at the same time, it is understandable that a radical procedure was advocated in view of the frequency with which these cases were associated with attacks of choking and strangulation. Keen[5] described sixty-seven cases of typhoidal perichondritis and stressed the importance of an early tracheotomy. According to his figures, the mortality without tracheotomy is 97 per cent and with tracheotomy 67 per cent. Hinsberg[8] gathered 365 cases of typhoidal perichondritis from the literature, covering the years 1819 to 1919, and of these 197, or more than half, died. Of 122 treated conservatively—that is, without tracheotomy, 107 died. Considering the cases of perichondritis of the cricoid as a separate group, the mortality would probably be 100 per cent. The prognosis was decidedly better when an operation was resorted to. Tracheotomy was done in 243, of which ninety died and the rest survived. However, in cases of perichondritis following grippe and in such laryngeal complications following infections of the neighboring tissues, the number of milder cases which occur and recover without operation (Case No. 1) is greater than the number of severe cases. The reason for this is that infections limited to the arytenoids occur more often in this etiologic group than they do in typhoid. But if necrosis of the cricoid occurs, the danger is just as great in cases of non-typhoidal origin as in those following typhoid.

It is only since 1904 that a systematic routine in the treatment of typhoidal perichondritis has been followed. This method was based on the principles promulgated by Schueller,[33] and he was the first to advocate an exact routine of treatment in these cases. His principles were: (1) Early exposure of the infective area through laryngotomy (laryngofissure), with removal of the sequestra and curetting of the abscess cavity. (2) In connection with the above, immediate dilatation to prevent stenosis. This systematic routine of treatment was first used by Hinsberg,[34] Gluck[35] and Ivanoff.[36] In 1905, Chevalier Jackson[6] published his observations on 360 cases of typhoidal origin. He advocates early tracheotomy.

It seems, therefore, that in the typhoidal cases a very radical procedure is often necessary because of the severity of these cases. Tra-

cheotomy and perilaryngeal incisions are often insufficient, and it is frequently necessary to do an exploratory laryngotomy with removal of the sequestra.

All later procedures advocated in the nontyphoidal infections are based on the experiences gained from the typhoidal cases. Hansberg,[37] too, recommends the radical procedures and advises not to delay tracheotomy and if possible to combine it with laryngotomy. This procedure is certainly advisable in complicated cases in which several of the laryngeal cartilages are involved, especially where the thyroid and cricoid cartilages are greatly affected; but, as already mentioned, milder cases occur in which only one cartilage, for example, the arytenoid, is affected and in these the infection is often self-limited and subsides without necrosis and abscess formation. Isolated abscesses of the vocal cords have been described by Chiari.[18] They usually evacuate themselves spontaneously or through a superficial incision. The same is true of the isolated abscesses of the epiglottis. Simple non-abscess forming inflammations of the other laryngeal cartilages have also been described, and these, too, heal without operation. It is, however, very difficult at times to determine whether such a mild infection is present or whether an abscess has already formed. The latter naturally requires operation in the majority of instances. We have reached the following conclusions from our studies of the literature and from our practical experience:

If dyspnea is present, an early tracheotomy should be done. In unilateral perichondritis limited to the arytenoid, it is safe to wait, but bilateral involvement very often causes suffocation and a tracheotomy is necessary to relieve it. Following this, the swelling of the cartilage usually recedes (Case No. 2). If the swelling does not recede in about a week, one must assume that there is an osteomyelitis of the arytenoid and treat it through a laryngotomy (laryngofissure). It is not necessary to remove the entire arytenoid, but the medial surface should be curetted out down into the marrow cavity, using a very sharp curette. Of course, if the entire arytenoid or its greater part is sequestrating, it is necessary to remove either the entire arytenoid or its greater part, which can be done without danger of a functional disturbance, provided that the lateral portion with its muscular attachments is left intact.

If there is widespread infection and destruction of the thyroid cartilage, simultaneously with the cricoid, we agree with those authors who advocate an extensive exposure of the larynx through laryngotomy and cricotomy. If one is convinced, however, that an infection of the thyroid cartilage alone is present, we suggest exposing the carti-

lage through an external incision, removing the diseased parts and thus draining the abscess. This procedure is very simple if fluctuation is already present over the outer surface of the thyroid cartilage. An opening into the cavity of the larynx should not be made without urgent reasons. Chronic submucous abscesses of the thyroid cartilage occasionally give but few signs. The diagnosis can be made at times from the appearance of the pyriform fossa (Kernan and Schugt[2]). We have seen such cases and have described them in detail at the end of this paper. The cartilage need not be involved in these cases. It is sufficient to expose the thyroid cartilage as shown in Fig. 18, and by means of a window resection of the cartilage expose the perichondrium. The abscess can then be incised and drained. A similar procedure can be followed in acute cases with necrosis of the thyroid cartilage. (See Case No. 3.)

In this case we found a necrosis with abscess formation of the upper border of the left thyriod lamina. The abscess had broken through and partly emptied itself into the pyriform fossa prior to operation. The question arose whether one should have waited after the abscess had evacuated itself. Some time after the first operation, however, a new area of necrosis developed from the anterior commissure and formed beneath the right lamina of the thyroid cartilage. This then necessitated a second operation to expose the right lamina. Because these abscesses are known to burrow downwards and lead to a mediastinitis, we recommend that, at the very least, the thyroid cartilage be exposed from the outside in these cases and all infected material be removed. If it seems necessary later on, a laryngotomy (laryngofissure) can always still be done.

There are, it is true, cases with other complications arising early in their course, which expire despite careful treatment of the laryngeal condition. This is particularly true in pneumonia and mediastinitis. Cases Nos. 5 and 6 are typical examples. These complications arise partly on a hematogenous basis and partly by burrowing of the abscess into the mediastinum. If such a complication does not arise within the first week, one can assume a good prognosis, provided that the local treatment of the laryngeal condition is adequate.

As previously mentioned, there is usually an osteomyelitis present in infection of the cricoid (O. Mayer[1]). One should, therefore, search for the infective area in the region of the ossified parts. If the entire cartilage is destroyed by the infection, the larynx collapses and a marked stenosis follows. Sometimes, though, retention of portions of the cricoid will prevent such a collapse. An endeavor should be

made to save as much of the cartilage as possible. We have not as yet seen a case of perichondritis or abscess limited to the cricoid alone. They are more rare than any other type. As to treatment, we must therefore again quote O. Mayer.[1] He recommends the following: After a tracheotomy which cannot be avoided in widespread involvement of the cricoid, and in conjunction with which cricotomy and laryngotomy should be done, a semicircular incision is made over the posterior half of the cricoid. This incision can also be extended laterally over the arch, and any abscesses present there can be opened. In younger patients, up to the twentieth year, one will find pus only in those parts of the lamina situated below the cricoarytenoid articulations. In early cases without necrosis an extensive incision and drainage of the infected marrow cavity will suffice. This should check the infectious process.

In chronic cases, the same semicircular incision over the posterior circumference of the cricoid should be made. Fistulæ, if present, are usually found in this line of incision. One should then search for loose sequestra, which are usually to be found in the lamina cricoidea. Any such sequestra are removed. Bony fragments which do not readily come away should be left behind, even if one feels that they are necrotic. So much for the bony portions of the cricoid. The purely cartilaginous portions are highly resistant to infection.

Cartilage which is in part still firmly adherent to its surroundings should not be considered necrotic and should therefore not be removed. Even cartilage that has been damaged can heal back into place. This holds for the various laryngeal cartilages. One should not be too radical, therefore, in treating the cricoid, especially not with the lamina, as its preservation is necessary to maintain the function of the larynx. All affected parts can be removed from the thyroid cartilage without hesitation, especially those cartilaginous portions that have become separated from their perichondrium. Almost the entire thyroid cartilage can be removed without impairing the function later on (Case No. 3). However, it is not advisable to free the perichondrium from the cartilage by means of an elevator without good reason, for cartilage which is firmly adherent is most always healthy.

REGENERATION.

Zarfl's[9] microscopic studies in a child of ten have shown that defects in cartilage caused by infections can rapidly be covered with connective tissue growing from the perichondrium, and that this tissue contains cartilage formed by the perichondrium in a relatively

short time. The perichondrium can form both cartilage and bone. These regenerations of destroyed cartilage are, however, only slight.

The formation of bone takes place chiefly in the granulation tissue, in which one finds islands of osteoblasts. The regeneration occurs chiefly in the vicinity of the osteomyelitic foci and takes place in the bones as well as in the periosteum. Concerning the regeneration of cartilage we also refer to the publications of Binder[38] and Genzmer.[39]

Amongst our cases there are two kinds: the acute[6] and the chronic.[2] It will be noted that they form a series beginning with a case which recovered almost spontaneously without any special treatment, and ending with cases which required extensive external drainage. One or two of these cases were fatal for lack of adoption, we contend, of that very method.

Report of Cases.

Acute Cases.

Case 1.—This was an example of laryngeal abscess which ruptured spontaneously and went on promptly to recovery. A man, 29 years old, was admitted to the Presbyterian Hospital, October 17, 1932, with a history that 36 hours before he had suddenly developed pain and a tender swelling on the left side of the throat. There was no difficulty in breathing. Swallowing was very painful.

Examination showed a swelling of the left aryteno-epiglottic fold completely concealing the vocal cord. There was some pus coming from the left pyriform fossa. The next day the swelling of the left arytenoid had subsided, and there was pus to be seen coming from an opening in the left glosso-epiglottic fossa (Vallecula). This would indicate that in this case the inflammation had spread towards the base of the tongue. Under conservative treatment, such as hot irrigations and sedatives, the abscess continued to drain and the patient was discharged from the hospital on the third day.

Case 2.—J. M., a man 38 years old, was admitted to the Presbyterian Hospital, February 20, 1933. This was the only one of our cases in which the suppuration appeared to originate primarily in the larynx. The attack was due to his choking on a piece of celery when at supper the night before admission.

This case is one degree greater in severity than number one. The respiratory difficulty was such that he required a tracheotomy immediately after admission. The laryngeal abscess required no direct treatment, as it ruptured spontaneously soon after the tracheotomy was done, and the patient promptly recovered.

At the time of his admission, the epiglottis and all of the laryngeal structures on the left were greatly edematous (Fig. 2). He was taken at once to the operating room and a tracheotomy was performed. The same afternoon a direct laryngoscopy was done. The epiglottis, the left aryteno-epiglottic fold, the left arytenoid, the left pharyngeal wall, the right ventricular band all participated in a great deal of swelling. There was a free discharge of pus into the lower pharynx, the source of which could not be exactly located. The right vocal cord and arytenoid were absolutely normal.

Fig. 1.

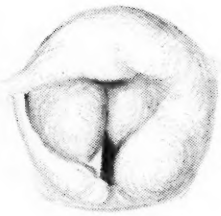


Fig. 2.

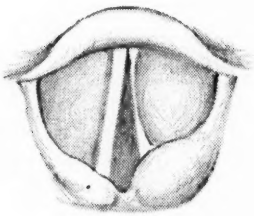


Fig. 3.

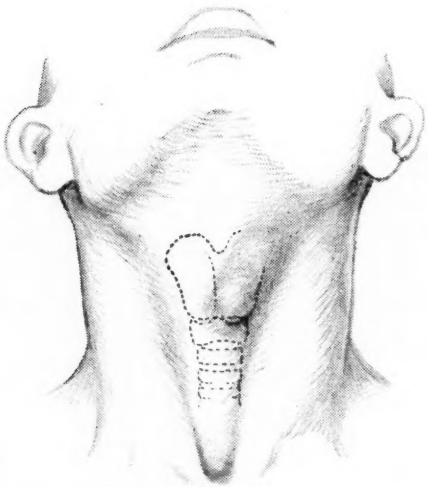


Fig. 4.

Fig. 1. The lamina (plate) of the cricoid cartilage showing the lateral bony nuclei on each side. The upper nuclei form near the cricoarytenoid articulations, the lower ones form near the cricothyroid articulations.

Fig. 2. Case 2.

Fig. 3. Case 3.

Fig. 4. Case 3. External swelling over the left thyroid plate and cricoid cartilage.

On account of the history of foreign body, x-rays of the larynx were taken. These films showed a marked swelling of the epiglottis and aryteno-epiglottic fold. The normal landmarks of the vestibule and ventricles were obliterated.

This man had no trouble whatsoever after his tracheotomy. His temperature gradually fell to normal. He was discharged without the tracheotomy tube the 13th day after his admission to the hospital.

CASE 3.—A. M., 35 years, physician, was admitted to Lenox Hill Hospital May 2, 1933. He had had tonsillitis a week previous, and for the previous two days pain on swallowing. Examination showed pharynx and both tonsils bright red and somewhat swollen. Larynx: Both arytenoid cartilages were very red, the left appeared to be swollen (Fig. 3). There was also a slight congestion and swelling of both ventricular bands, more marked on the left side. A moderate tenderness on pressure could be made out around the upper border of the left thyroid plate, together with a slight swelling. Blood count 36,000 white, mostly polynuclear cells. Temperature rose to 106° around midnight. The patient expectorated a small amount of thin mucopus from the region of the larynx, but no fistula or opening of an abscess cavity could be seen with the laryngeal mirror. Some of the secretion was collected and the culture showed streptococcus hemolyticus. Wassermann reaction was negative. X-rays of the chest revealed no tuberculosis.

May 3. The swelling of the left arytenoid and left ventricular band increased. There was also a distinct swelling of the left side of the epiglottis. Voice very hoarse. Tenderness over the left thyroid plate was also more pronounced today and a slight swelling was now present, reaching from the upper border downwards to the cricoid and crossing the midline (Fig. 4).

May 4. Left arytenoid appeared to be fixed and more swollen. The left pyriform fossa was considerably swollen (Fig. 5). Patient now coughed up more pus from the larynx. The tenderness and swelling on the outside was much more pronounced and reached further down below the cricoid cartilage. We had the impression that the process might be going down into the mediastinum. Temperature 102° in the afternoon. No chills. Patient could swallow only with great difficulty and severe pain.

May 5. Direct laryngoscopy. A large amount of pus was coughed up from the throat while the tube was introduced. No abscess cavity could be seen. External operation: Incision over the left side of larynx (Fig. 6). The left thyroid plate was exposed and showed a soft necrotic area at the upper border. This necrotic tissue was removed with a curette and an abscess cavity could be seen leading towards the left pyriform fossa. About two or three ccm. of green pus was evacuated. A rubber tube was inserted into the cavity and later on with the laryngeal mirror it could be seen protruding into the pyriform sinus.

May 6, 7, 8. The patient's temperature gradually came down. Feeding was given through an Einhorn tube. Ten ccm. of antistreptococcus serum was given. The swelling in the larynx markedly decreased.

May 12. Temperature normal. Voice clearer. Patient could now swallow without difficulty. Einhorn tube removed.

May 17. The swelling of the epiglottis disappeared. Left arytenoid only very slightly swollen and movable. Left pyriform fossa normal.

May 27. Left ventricular band more swollen. Temperature slowly rising.

June 2. Temperature 100° in the afternoon. Left arytenoid more swollen.

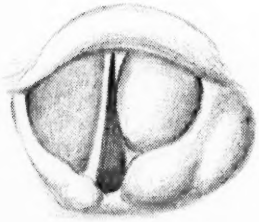


Fig. 5.

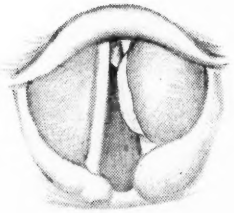


Fig. 7.

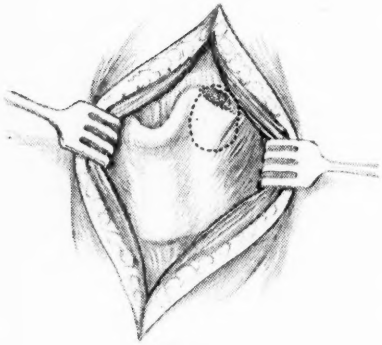


Fig. 6.

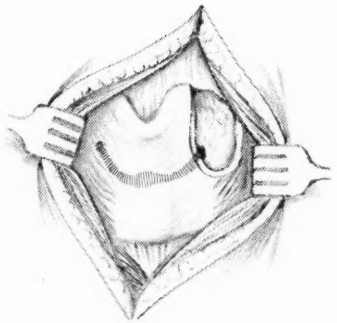


Fig. 8.

Fig. 5. Case 3. Swelling of left side of epiglottis, left ventricular band, left arytenoid and pyriform fossa.

Fig. 6. Case 3. Thyroid cartilage exposed. Left upper border necrotic. Dotted line indicates the size of abscess found behind the left thyroid plate.

Fig. 7. Case 3. Swelling of left ventricular band and prolapse of left laryngeal ventricle together with swelling of left arytenoid. Note also the subglottic swelling in the anterior commissure of larynx indicating a process in the junction of both thyroid plates.

Fig. 8. Case 3. Second operation. Reopening of wound. On the left side is shown the area of necrotic cartilage removed at the first operation (Fig. 6). A fistulous tract leads from here across the mid-line towards the right arytenoid cartilage. The thyroid cartilage covering this fistula appeared to be soft and necrotic and was removed leaving only the upper and lower border.

June 7. Direct laryngoscopy: Swelling and prolapse of the left laryngeal ventricle. Temperature stayed between 100° and 101°. External wound had healed.

June 10. Pain on swallowing. Tenderness over the outside scar. Left ventricular band swollen. Prolapse of left ventricle. There was now a subglottic swelling in the anterior commissure (Fig. 7).

June 12. Re-opening of the external incision (Fig. 8). A necrotic area was found over the midline of the thyroid cartilage and a fistulous tract in the inner perichondrium and submucosa was seen crossing the midline and leading towards the right arytenoid cartilage. A large part of the cartilage was removed from the midline and from the right thyroid plate. The fistulous tract was split open and a small rubber tube inserted. The underlying mucosa was left intact.

June 13. Temperature was down. Both ventricular bands much swollen. Prolapse of both laryngeal ventricles.

June 19. Swelling in the larynx decreasing. External wound healing. Temperature normal. Voice clearer.

June 22. Both vocal cords could be seen now on phonation. There was still a prolapse of both laryngeal ventricles (Fig. 9). The external wound was healing but still open. The patient left the hospital for Europe.

The patient was seen again three months later, at which time the external wound was closed. The voice was slightly hoarse at this time. Laryngeal examination showed only moderate redness of both cords.

CASE 4.—B. F., a girl 13 years old, was admitted to the Harbor Hospital, May 30, 1933. Her illness began five days before with what seemed to be follicular tonsillitis. The day before her admission to the hospital she was improved, with less soreness of throat and lower temperature.

The day of her admission to the hospital she became much worse. There was difficulty in swallowing, dyspnea, and her temperature rose to 104°. Examination of the larynx showed a swelling which seemed to involve the whole right side—that is, the right side of the epiglottis, the aryteno-epiglottic fold and right ventricular band (Fig. 10).

The night of her admission, a direct laryngoscopy tube being used, incisions were made in the tip of the epiglottis and along the crest of the aryteno-epiglottic fold (Fig. 10). A small amount of pus was released. The next day her temperature fell to 100° in the afternoon. The dyspnea was relieved, and the swelling in her larynx was considerably less. Swallowing was still painful.

The next morning her temperature rose to 104° and then fell rapidly to 101°. In spite of the rise of temperature in the morning, her symptoms were much improved. This was on the fifth day after admission to the hospital. For the next two days she made steady progress.

Examination at this time showed that the swelling of the epiglottis and the aryteno-epiglottic fold had disappeared. There was, however, swelling from the right pyriform fossa (Fig. 11). In spite of the persistence of this swelling, the patient was very comfortable; there was no difficulty in swallowing or breathing; the temperature, however, continued elevated about 101°.

On the morning of June 7th, after a very comfortable night, she woke up with a great deal of pain in her throat and a large tender swelling exteriorly just

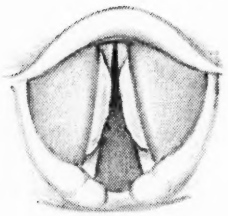


Fig. 9.

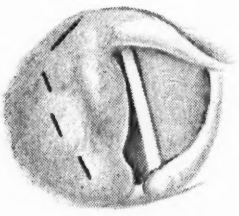


Fig. 10.

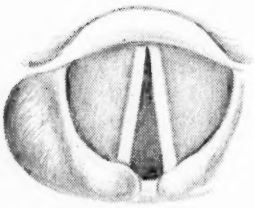


Fig. 11.

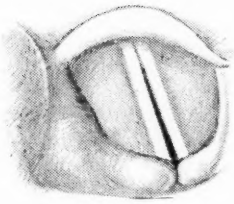


Fig. 12.

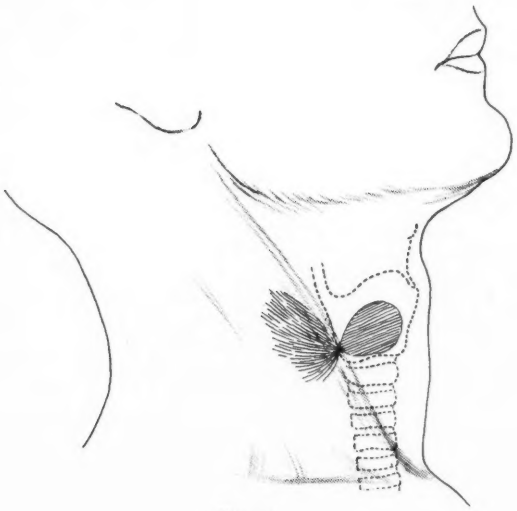


Fig. 13.

Fig. 9. Case 3. Prolapse of both laryngeal ventricles.
Fig. 10. Case 4.
Fig. 11. Case 4. Swelling of right pyriform fossa.
Fig. 12. Case 4. The swollen right pyriform fossa and right aryepiglottic fold form one large mass. This mass together with the whole side of the larynx are pushed over to the left by an extensive swelling of the right paralyngeal space, containing the abscess cavity.
Fig. 13. Case 4. Side view. Showing the abscess in the larynx and the point where it had broken through into the paralaryngeal space, pushing the sternomastoid muscle laterally and pressing the right laryngeal plate to the left (Fig. 12).

below the angle of the jaw. Laryngeal examination now showed a large mass involving the right aryepiglottic fold and pyriform fossa. This mass was pushed over to the left side by a swelling of the right lateral pharyngeal wall (the para laryngeal space) (Fig. 12). The appearance of the larynx together with swelling on the outside was evidence that the pus had made its way to the outer side of the larynx to the sheath of the great vessels (Fig. 13). It was now necessary to perform an external operation.

An incision was made along the anterior border of the sternomastoid muscle, and deepened to the sheath of the vessels. Pus was found in or beneath the sheath of the great vessels on the external surface of the inferior constrictor of the pharynx. The abscess was widely opened and drains inserted. Thereafter, the patient made steady improvement and was discharged from the hospital exactly three weeks after the first opening of the abscess through the mouth.

It is the contention of the authors that if the abscess in the pyriform fossa had been opened in time by a window resection through the thyroid cartilage, which we will describe later on, the illness could have been considerably shortened and the threat of mediastinitis entirely averted. We conceive the route of the pus as having been from the pyriform fossa about the posterior edges of the thyroid cartilage (Fig. 13) into the region of the great vessels.

CASE 5.—A. T., a woman, 36 years old. In this case the pus took a somewhat similar route, but, unfortunately, was not operated on before it had passed down the neck and reached the mediastinum. This patient was admitted to the Presbyterian Hospital on February 19, 1926, with a history of an illness which began with a sore throat of five days' duration. It was accompanied by headaches, malaise, high fever, very painful swallowing.

The diagnosis was follicular tonsillitis and acute laryngitis.

There was diminished resonance and breath sounds over the bases of both lungs.

The next day she developed considerable difficulty in breathing. Examination of her throat showed a negative pharynx and considerable swelling of the epiglottis, the left arytenoid, the left aryteno-epiglottic fold and both ventricular bands (Fig. 14). The dyspnea increased severely and the next day necessitated an incision of the abscess. Pus was found in the swelling of the arytenoid and anteriorly in the epiglottis. This opening of the abscess relieved her dyspnea but not her illness as a whole. Her fever continued high; she developed signs of pneumonia and mediastinitis and died the tenth day after her admission to the hospital.

The thing of immediate interest to us is the route of the pus from the larynx to the mediastinum. The autopsy showed that it had ruptured through the cricothyroid ligament and then had passed down the neck a short distance and crossed to the right side between the trachea and the esophagus and then down into the mediastinum (Fig. 15). There was a small tract also passing down the left side to the mediastinum, but the tract down the right side was much larger.

This case is interesting as showing the dangers in these cases once the pus has reached the deep spaces of the neck. As the matter turned out, the patient could not have been saved, as she had in addition to the mediastinitis, a septic pneumonia and empyema.

CASE 6.—J. Z., a man, 27 years old. This case illustrates with what caution one should undertake the incision of abscess of the larynx from above when there is great swelling and distortion of the parts.

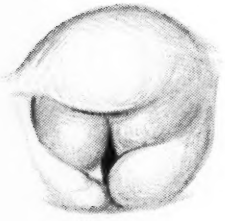


Fig. 14.

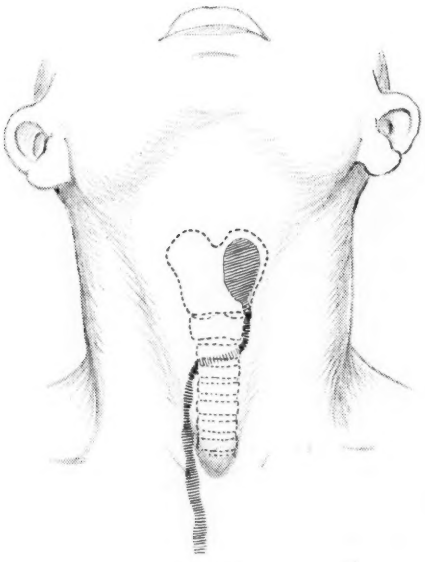


Fig. 15.

Fig. 14. Case 5.

Fig. 15. Case 5. The abscess in the larynx behind the left thyroid plate has broken through and burrowed downward behind the trachea into the right side of the mediastinum.

This patient was admitted March 3, 1931, to the Presbyterian Hospital, with a history of having had pain in his throat and difficulty in breathing and swallowing for three days.

Examination showed considerable swelling in the region of the arytenoepiglottic fold. The man was in great distress and had considerable difficulty in breathing.

He was taken at once to the operating room. The larynx was exposed with a direct laryngoscope, and an incision made into the most prominent part of the swelling. This released pus, but almost immediately there was a very profuse hemorrhage which in addition to the strangulation due to the swelling in the larynx, almost resulted in immediate death from asphyxia.

An immediate tracheotomy was done. Then, to control the hemorrhage, an incision was made along the anterior border of the left sternomastoid muscle and deepened to the sheath of the vessels. There was a tremendous hemorrhage from this region, the origin of which, on account of its volume, could not be ascertained. This hemorrhage was controlled by packing.

The subsequent history of this patient was tragic. On March 17th, two weeks after his admission, during a coughing attack, he had a rather profuse hemorrhage both from his mouth and from the external wound. It was then decided to make an attempt to find the bleeding point in the neck. This was

successfully accomplished three days later by making a long incision at the anterior border of the left sternomastoid muscle and exposing the great vessels. The vein and artery were found to be bound together in a mass of inflammatory tissue. The bleeding point was located but such was the denseness of the tissue about, that it was finally necessary to pass a ligature about the internal jugular vein and the common carotid artery both above and below the bleeding point in order to control the hemorrhage. This was done. The patient seemed to suffer no bad effects from the ligation of the common carotid artery. However, there was extensive suppuration in the wound of the neck. The pus made its way into the mediastinum as low as the eighth thoracic vertebra; also it was found impossible to prevent aspiration of the pus from the wound in the neck via the tracheotomy wound. So the patient ultimately developed an abscess of the right lung, which drainage was not successful in relieving, and he died of sepsis three months after admission to the hospital.

Chronic Cases.

CASE 7.—Miss A. R., age 14, was admitted to Lenox Hill Hospital, October, 1930. She complained of hoarseness of several months' duration. She stated that she had a cold a few months ago, and had had increasing hoarseness since then. There was no pain on swallowing, and no dyspnea. In general, she felt well. Laryngoscopic examination revealed edematous swelling of the entire left pyriform fossa and similar thickening of the left ventricular band, so that the vocal cord was almost entirely hidden (Fig. 16). However, the function of the vocal cords was apparently normal. General examination gave negative results. Roentgenograms of the lungs and lateral views of the larynx revealed nothing. The Wassermann test was negative. With the aid of a mirror and a guarded laryngeal knife, an incision was made into the mass in the pyriform fossa, releasing about 5 ccm. of thick yellow pus and with some immediate relief of hoarseness. Bacteriologic examination showed streptococcus hemolyticus. The patient was seen at weekly intervals, and it was noted that the abscess in the pyriform fossa filled repeatedly. As a result, the abscess was reopened about eight times, sometimes indirectly with a bent laryngeal knife and on several occasions through the Jackson laryngoscope with a straight knife, and at each instance the same amount of pus was released. During the second month following the last incision, the patient's voice was clear and the swelling was slight. In May, 1931, the patient returned, and the entire pyriform fossa was involved with accompanying swelling of the left ventricular band and marked hoarseness. Operative procedure will be described below.

CASE 8.—Mr. C. B. O., on Oct. 21, 1925, complained of pain in the throat and difficulty in swallowing for several weeks. Examination of the larynx showed a swelling of the left aryepiglottic fold and left pyriform fossa. A diagnosis of laryngeal abscess was made, and incision through the direct laryngoscope released a large amount of foul pus. Bacteriologic examination showed staphylococcus aureus. The swelling, however, did not entirely disappear and the incision had to be repeated, in all about eight times, on each occasion the same amount of pus being evacuated. The patient was seen regularly and the swelling seemed much smaller, but occasionally it would increase in size. At times a small amount of pus could be seen oozing out of a small fistula. In June, 1926, the patient returned with the same symptoms as in the beginning, and examination revealed the same area to be much swollen, involving the left arytenoid cartilage.

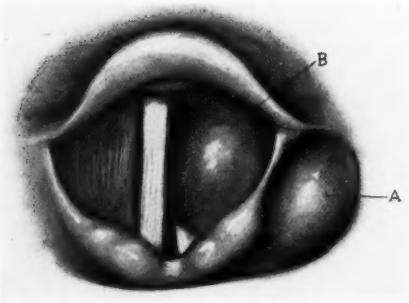


Fig. 16.

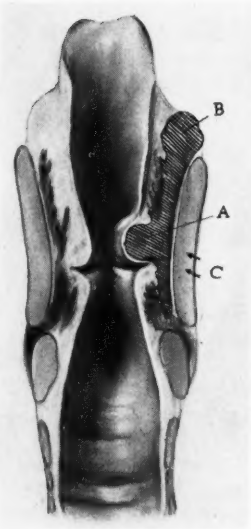


Fig. 17.

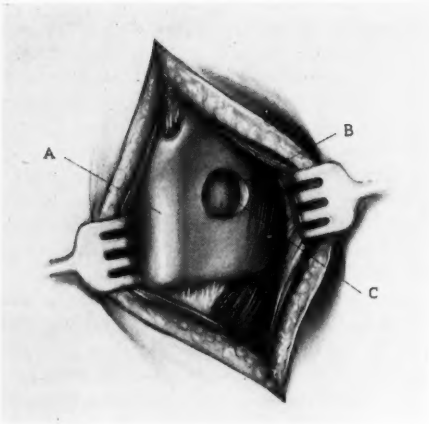


Fig. 18.

Fig. 16. Cases 7 and 8. Abscess of the larynx pointing into the pyriform sinus *A* and into the ventricular band *B*.

Fig. 17. Cases 7 and 8. A section through the larynx to show the supposed extension of the abscess: *A*, the abscess pushing into the lumen of the larynx. *B*, the abscess bulging into the pyriform fossa, and *C*, arrows showing the site of the window made through the cartilage in order to approach the abscess.

Fig. 18. Cases 7 and 8. *A*, thyroid cartilage. *B*, window in the thyroid cartilage to approach the abscess through the inner perichondrium, *C*.

It is evident that Case No. 7 and No. 8 resemble each other in the laryngoscopic picture as well as in their clinical course. We were sure of the diagnosis of laryngeal abscess and also realized that superficial incisions would not result in a cure. We, therefore, had to assume that the abscess was situated more deeply, apparently in the inner part of the larynx. We also assumed that the abscess had formed in the submucosa of the thyroid cartilage in the interior part of the larynx and had pointed upward, into the pyriform fossa (Fig. 17). We decided to attempt an approach and drainage of the abscess from the outside by creating a window in the thyroid cartilage. The same procedure was used in both cases. The thyroid cartilage was exposed as Fig. 18 shows, an opening the size of a one cent piece made with a sharp curet. The perichondrium of the interior of the larynx bulged out into the opening and was fluctuant. An incision into the perichondrium was made and about 10 to 15 ccm. of thick creamy pus was released in both cases. This proved that the abscess was situated between the inner perichondrium of the thyroid cartilage and the submucosa of the larynx. A rubber drain was inserted. The wound was kept open for several weeks by means of a drain and was completely healed within the next three weeks.

Both patients were seen one year after the operation, and the larynx was found to be entirely normal with a scar on the outside over the thyroid cartilage.

SUMMARY.

Laryngeal abscess is a rare disease. It occurred more frequently in former years as a complication of typhoid fever. Many such cases were published at that time, and the present knowledge about the diagnosis and treatment of abscess of the larynx is largely based on the experience that those authors had during the typhoidal epidemics.

In recent years abscess of the larynx is seen less frequently and at present is chiefly seen as a complication following grippe or in connection with an acute tonsillitis and pharyngitis. Other diseases causing such laryngeal complications are scarlet fever, measles, septicemia, puerperal sepsis, erysipelas, gonorrhea, syphilis.

The traumatic form (foreign body) of laryngeal abscess is the only one that has its origin in the larynx itself. Other cases result from external trauma to the larynx. Presence of tumors in the larynx (carcinomata) can cause abscess formation by breaking down and becoming infected.

Pain is the first symptom to appear. Hoarseness is seldom absent. Dyspnea is often present and one of the most dangerous symptoms. The most rarely affected cartilage is the cricoid cartilage. If there is abscess formation in the arytenoid or epiglottis, spontaneous evacuation can take place. Inasmuch as the laryngeal cartilages in the adult are all ossified to a large degree a real osteomyelitis can occur in any of the laryngeal cartilages. A moderate swelling of the arytenoid as a sign of perichondritis can subside without abscess formation. Then there sometimes remains a thickening and fixation of the arytenoid

cartilage. In other cases the cartilages or parts thereof are sloughed out as sequestra.

In the thyroid cartilage the infection sometimes appears as a submucous laryngitis and then goes on to a perichondritis and abscess formation. In some cases the infection breaks through the cartilage and appears externally. In abscess of the thyroid cartilage, the pyriform fossa plays an important part in the diagnosis. In such cases the pyriform fossa bulges upwards because the upper and posterior portions of the thyroid cartilage form an unyielding wall externally.

Abscesses of the larynx, especially of the thyroid cartilage, have the tendency to burrow downwards and lead to mediastinitis. In other cases a metastatic pneumonia or lung abscess may develop.

On the other hand, there are abscesses which are limited to the submucosa, but in which the infection is quite extensive. The abscess can be present for a considerable time in the submucosa without penetrating the deeper layers, the perichondrium or cartilage.

Many differences of opinion exist as to the treatment of laryngeal abscess. On one hand are those authors advocating conservative treatment, and on the other those advocating the most radical surgical procedures. Since most of the cases occurring in former years resulted as complications of typhoid and since in this infection several of the laryngeal cartilages were often affected at the same time, it is understandable that a radical procedure, such as tracheotomy and laryngofissure, was advocated in view of the frequency with which these cases were associated with attacks of choking and strangulation.

However, in cases of perichondritis and abscess formation following grippe or the other nontyphoidal infections, the number of milder cases which recover without operation is greater than the number of severe cases.

The textbooks usually mention that exclusive of a tracheotomy and superficial intralaryngeal incisions the most common method to evacuate abscesses of the larynx is by doing a laryngofissure, especially when the abscess is located in the inside of the larynx. We do not agree with these authors and believe that many such cases can be treated with more conservative operations. In this paper an original method ("window resection of the cartilage") is described, which we employed to evacuate submucous abscess of the thyroid cartilage without doing a laryngofissure. Eight cases are described in this paper. They form a series beginning with a case which recovered almost spontaneously without any special treatment, and ending with cases requiring extensive external drainage.

30 East 40th Street.

BIBLIOGRAPHY.

1. Mayer, O.: 1. Beitraege zur Behandlung der Perichondritis laryngea auf Grund eigner klinischer u. histologischer Untersuchungen. Zeitschr. f. Hals-Nasen-Ohren, 28:309-370, 1931.

2. Acute Perichondritis. Wien. Klin, Wochenschr., 1:223, 1932.

3. Perichondritis laryngea: Neue Gesichtspunkte. Monatsschr. f. Ohrenheilk., 66:242, 1932.

2. Kernan, John D., and Schugt, Henry P.: Primary submucous laryngeal abscesses. Arch. of Otolaryn., 17:22-29 (Jan.), 1933.

3. Kuttner, A.: Das Larynxoedem und die submucoese Laryngitis. Virchow's Archiv. f. path. Anat., 139:117, 1895.

4. Seiferth, L. B.: Ueber die Perichondritis u. Chondritis des Kehlkopfes. Zeitschr. f. Hals-Nasen-Ohrenheilk., 22:102-127, 1928, 1929.

5. Keen, W. W.: 1. On the Surgical Complications and Sequels of Continued Fevers. Smithsonian's Miscellaneous Collections. Wash., 1877.

2. The Surgical Complications and Sequelæ of Typhoid Fever. Philadelphia, 1898.

6. Jackson, Chevalier: The Larynx in Typhoid Fever. A. J. of Med. Sci., 130:845, 1905.

7. Rieser: The Laryngeal Complications of the Typhoid Fever. N. Y. Med. Journ., Feb. 29, 1908.

8. Hinsberg, V.: Die Kehlkopferkrankungen im Typhus abdominalis in Madelung: Die Chirurgie des Abdominal-typhus. Neue Deutsche Chirurgie, 30:11, Stuttgart, 1923.

9. Zarfl, Max: 1. Ueber Klinik u. Anatomie der Perichondritis laryngea im Kindesalter. Zeitschr. f. Kinderheilk., 36:242, 1923.

2. Perichondritis laryngea im Saeuglingsalter. Wien. Med. Wochenschr., 33:34:1438-1439, 1920.

10. Imhofer, R.: 1. Larynx Abscesse. Zeitschr. f. Ohrenheilk., 76:36, 1917.

2. Metastatische Larynxabszesse im Verlaufe einer Pyamie. Intern. Zentralbl. f. Laryn. u. Rhin., 31:116, 1915.

11. Birkett, H. S.: Perichondritis of the Larynx. Trans. of the Amer. Laryn. Assoc., p. 185, 1896.

12. Forschner, L.: Larynxphlegmone bei Mittelohreiterung. Wien. Laryngorhinol. Gesellsch., April 1, 1930.

13. MacMahon, H. E.: Ueber eine seltene u. schmer erkennbare Folge der Tonsillitis in Form einer starren, stenosierenden Kehlkopfphlegmone. Zeitschr. f. Hals-Nasen-Ohrenheilk., 2tes Heft., 22:247, 1932.

14. Wiehe, C.: Ueber die Tonsilla sinus pyriformis: Ein Beitrag zur Aetiologie des akuten Larynxoedems. Zeitschr. f. Hals-Nasen-Ohrenheilk., 30 Heft., 2:235, 1931.

15. Türck, L.: Klinik der Krankheiten des Kehlkopfes u. der Luftroehre. Vienna, 1866.

16. Von Schroetter, L.: Vorlesungen ueber die Krankheiten des Kehlkopfes. Vienna W. Braumueller, p. 99, 1892.

17. Hajek, M.: Heymann's Handbook, Vol. 1, 1898.

18. Chiari, O.: 1. Chirurgie des Kehlkopfes u. der Luftroehre. Stuttgart, Ferdinand Enke, 1916.

2. Ueber primaere acute Entzuendungen des submucoesen Gewebes des Kehlkopfes. Wien. Klin. Wochschr., 10:109, 1897.

19. V. Heymann, Paul: Handbuch der Laryngologie und Rhinologie, Vienna, A. Hoelder, 1898.

20. Levinstein, Oswald: Pathologie u. Therapie der Epiglottis abscedens. Zeitschr. f. Laryn., 8:310, 1916-1919.

21. Jackson, Chevalier, and Coates, G. M.: The Nose, Throat and Ear and Their Diseases. W. B. Saunders, Phila., p. 844, 1929.

22. Hecht: Zur Lehre von den Kehlkopfabszessen. Thesis. Wuerzburg.

23. Tobold: Larynxabszess. Berlin. Klin. Wchnschr., 1:39, 1864.

24. Verweyen, Theodor: Ueber Larynxabszess. Wuerzburg-Becker, 1881.

25. Cruveilhier: Anatomie pathologique du corps humaine. Paris, T. B. Bailliere, Vol. 1, Book 2, Fig. 1, 1829.

26. Descortes, Renè: Les Abscès laryngés chez L'enfant. Thesis, Paris, 1912-13.

27. McIntosh, R., and Nicol, K. D.: Abscess of the Larynx in Infants. J. A. M. A., 90:2095, 1928.

28. Brueggemann, A.: Perichondritis and Thyroiditis. Deutsch. Med. Wchnschr., 46:97, 1920.

29. Békarian, Ajot: L'abscès froid prélayngé. Thesis, Paris, 1907-1908.

30. Pieniazek, P.: Die Verengerungen der Luftwege. Vienna, p. 140, 1901.

31. Albrecht: Perichondritis bei einem Kinde. Internat. Zentralbl. f. Laryn., 28:386, 1912.

32. Chievitz: Ueber die Verknoecherung der Kehlknopfknorpel. Anat. u. Physiol., p. 302, 1882.

33. Schueller, M.: Die Tracheotomie, Laryngostomie u. Exstirpation des Kehlkopfes. Deutsche Chirurgie, 1880, Lieferung 37.

34. Hinsberg, V.: Ueber die chirurgische Behandlung der: Perichondritis laryngis. Zeitschr. f. Ohrenheilk., Vol. 62, 1911.

35. Gluck, Th.: Vorschlaege u. Kasuistischer Beitrag zur Chirurgie der oberen Luft- und Speisewege. Zeitschr. f. Laryn. Rhin., Vol. 1, 1909.

36. Iwanoff, Alex: Laryngostomie bei Perichondritis des Kehlkopfes. Zeitschr. f. Laryn. Rhino., 2:241, 1910.

37. Hansberg: "Die Laryngofissur." Im Handbuch der speziellen Chirurgie des Ohres u. der Luftwege. Katz, Preysing, Blumenfeld, Wuerzburg, 4:185, 1912.

38. Binder, Alfred: Zur Frage der Knorpelregeneration beim Eerwachsenen. Beitraege z. Path. Anat., u. z. Allgem. Path. Supplement 7, p. 515.

39. Genzmer: Ueber die Reaktion des hydlinen Knorpels auf Entzuendungen. Virchow's Arch. f. path. Anat. u. Physiol., p. 67, 1876.

LXXXIV.

MÉNIÈRE'S SYMPTOM COMPLEX: MEDICAL TREATMENT.*

A. C. Furstenberg, M. D.,

F. H. Lashmet, M. D.,

Frank Lathrop.

Ann Arbor.

Since the classical description of Ménière's disease was given to medical science in 1861, much speculation has occurred and many pertinent recommendations have been made, referable to the treatment of this symptom complex. For many years the therapeutic management of this malady has been a controversial matter and one in which a confusing number of remedies have been offered, based chiefly upon the hope that they might in some manner influence some of the many hypothetical, pathologic changes which are assumed to exist.

The sum total of these endeavors has been of little value to the patient and the causes of failure are clearly those of trial and error. Assuming, for example, that an increased secretion of cerebrospinal fluid is responsible for pressure upon the eighth nerve, the lumbar puncture is advocated. Or, considering the pathogenesis associated with the sympathetic and parasympathetic nervous systems, such drugs as pilocarpin, atropin and adrenalin are administered because of some pharmacologic action upon nervous tissue. Recent studies in the field of endocrinology which suggest a possible relationship between a glandular dysfunction and epilepsy, the latter seeming to be akin to Ménière's disease, have occasioned an investigation of the patient's calcium metabolism and prompted the use of thyroid, parathyroid and pituitary substances. Quinin, for its specific effect upon the auditory nerve, potassium iodid, for its empirical values, bacterins and vaccines for what they may be worth, and finally, the application of a seton to the nape of the neck, are the remaining therapeutic measures available for trial.

Theoretically, some appear to have merit. We do not doubt the value of lumbar puncture in certain cases of vertigo. The effect of

*Presented before the annual meeting of the American Otological Society, Atlantic City, June 7, 1934.

free catharsis and pilocarpin sweats has properly given rise to much enthusiasm in others. These measures, conceived in good faith, have been promoted by an honest conviction that they have cured certain individuals of an intractable vertigo and that they are capable of doing the same for others. Yet who has not applied this large group of therapeutic possibilities only to end with a feeling of futility and a sense of despair. The lack of sustained practicability of medical therapy is constantly in evidence and, lest our claims appear presumptuous, may I add that the conclusions derived from our own studies are probably no exception to this fact.

There are other obvious reasons for the bewildered and futile state of medical therapy in Ménière's symptom complex. Consider, for example, the enormous variety of vertigo syndromes to which medical treatment has been applied. The tendency to create a disease entity by grouping a number of cases having one symptom in common is one of the temptations of medical practice. Vertigo lends itself admirably to this evil. Every conceivable form of dizziness appears to have been classified as a disease and treated as such. Failure to designate disease entities properly and apply well directed treatment has made a correlation of results impossible. There exists, therefore, no uniformity of opinion in respect to the merits of the innumerable therapeutic measures which have been employed.

The point of the foregoing is that many otologists have made their own classification of Ménière's symptom complex. They have done so whimsically and without consideration of the scientific facts upon which a clinical entity is developed. It has been a term loosely applied and much abused. So much has this been true that many authors have urged the abandoning of the personal reference and the acceptance of a more precise nomenclature. No matter what justification may exist for such a change, it remains a fact, nevertheless, that the great French clinician described a syndrome definitely related to the acoustic nerve and one which has borne his name for nearly three-quarters of a century. I strongly suspect that future generations of our profession will continue to accord it this personal reference.

Consider the syndrome which is typically Ménière's symptom complex: It embodies well defined clinical manifestations which have been exhaustively treated in our medical literature and are well known to every student of otology. A simple glossary will suffice to enumerate and place on record the symptoms which characterized the disease as it was treated in the following studies: A violent attack of vertigo in a patient with deafness; occasionally impaired cochlear function on both sides, but more profound in the ear to which the

symptoms are referred; nausea, vomiting and spontaneous nystagmus in the severe seizures; persistent tinnitus—often bilateral—increased during the paroxysm; vestibular tests of little significance—usually within the limits of normal; a pathetic picture of total disability in an individual suffering from a vertigenous attack, or living in the terror of an impending one.

The pathological changes responsible for the capricious symptoms of this syndrome still remain a mystery. They have been the subject of numerous studies, varying from conscientious research in the pathological physiology of the auditory mechanism to skillful adaptations of morbid processes to meet the clinical needs. They comprise voluminous works in our medical literature, quotations from which at this time would merely constitute a repetition of medical history. Suffice to say for those who hold that the pathological changes are in the internal ear, that statistical evidence indicates that the destruction of the otic labyrinth in a few cases has not been followed by relief of symptoms. To those who prefer the theory of morbid changes to the auditory nerve, I might add that microscopic examination of sections from the acoustic nerve in eleven cases failed to demonstrate pathologic alterations in the tissue submitted for study. In nine of the cases, relief immediately followed section of the nerve. In two of the cases the symptoms are still present after periods of two and four years. Dr. Konstantin Löwenberg, pathologist in our Psychopathic Department, in a recent study of autopsy material from a fatal case of tumor of the acoustic nerve, has made an extraordinary observation in the discovery of a normal nucleus in the presence of a completely degenerated auditory nerve. This amazing phenomenon, if sustained by further studies, would suggest an innervation of the nucleus from the opposite side and offer another site in the auditory system where the influences of pathologic changes might be felt. Obviously a nuclear lesion under such conditions would not be affected by section of the auditory nerve, and herein may lie a possible explanation for the failure, in rare instances, to obtain immediate and complete relief following severance of the acoustic nerve.

It is doubtful, therefore, if the clinical manifestations of Ménière's symptom complex can be definitely ascribed to any isolated part of the auditory tract. Rather is it more accurately assumed, perhaps, that any or all of its constituents may participate in the changes which give rise to a typical attack. There is some proof for this assumption in the observations already recorded, while the conclusions derived from our own studies do not tend to detract from this belief.

The theory which gave rise to the following investigation was first suggested to me by Dr. Samuel Kopetzky. To him, I gratefully accord full credit for the inspiration which led me into a most fascinating study. In September, 1933, he asked me the following questions: "What class of individuals have vertigo? Why are dizzy attacks frequently seen in patients with cardio-vascular-renal disease?" "The answer," he continued, "lies in some disturbance in the metabolism of water which gives rise, perhaps, to a water-logged condition of the static labyrinth. Why don't you study water balance in these patients?"

In acting upon Dr. Kopetzky's suggestion, I reviewed the medical literature for all available references on the subject. The only technical studies of interest were those of Dida Dederding of Copenhagen,[1] in 1929 and 1931, in which she advanced the idea that the symptoms in Ménière's disease were due to a disturbed metabolism of water.

She observed that patients were more likely to have an exacerbation of symptoms, when there was evidence of retention of body water. Furthermore, these patients were improved when they eliminated a large amount of water, as by profuse perspiration (pilocarpin) or polyuria (diuretics, chiefly salyrgan). She investigated this question by comparing the intake of fluid with the amount of urine excreted, and correlating the difference between them with changes in body weight. From the data so obtained she concluded that the symptoms of Ménière's disease were due to "an accumulation of water in the body," and that this fluid was probably "extra-cellular edema." From the data submitted there is no doubt that some justification for these conclusions exists, but there are certain aspects of this investigation which prevent complete acceptance of the explanation offered by Dederding.

First, edema, or the "accumulation of water in the body," is not water alone, but a solution of electrolytes, chiefly sodium salts, and water. Both of these substances, salts and water, are so dependent upon each other in maintaining isotonicity with the other body fluids that it is impossible to consider either alone. For example, when body water is lost by perspiration induced by pilocarpin, or by diuresis induced by salyrgan, a very definite amount of sodium salts is also lost in the process. One might ask then, if the relief of symptoms in Ménière's disease is not due more to the loss of sodium salts than to the water in which they are eliminated. Dederding does not consider this a possible explanation, although her data would support

such a conclusion just as well as it does her assumption that water is specifically concerned.

Second, the method by which Dederding and her associates measured water exchange was so inaccurate that quantitative statements regarding the latter are impossible. If an accurate statement of water exchange is desired, the amount of water "into" the body from every "source" and the amount of water "out" of the body at every "exit" must be determined. As will be seen, only a small fraction of both the water "into" and "out" of the body was determined by Dederding.

Table 1 lists the various items of water "into" and "out" of the body and gives the average amounts of each as obtained under ordinary conditions with the normal subject. Examination of this table reveals several interesting facts.

TABLE I.—WATER EXCHANGE—BALANCE.

AVAILABLE WATER.	Grams.	EXCRETED WATER.	Grams.
1. Exogenous		3. Urine	1000
A. Diet	900	4. Stool	200
B. Water as such	1100	5. Vapor	1000
2. Endogenous			
A. Oxidation	200	Total	2200
B. Preformed	0		
Total	2200		

The water contained in the solid food of the diet is a relatively large amount. The quantity of water derived from the oxidation of protein, fat and carbohydrates, although relatively small, is not slight enough to be neglected. The "preformed" water which is that water released from body tissue when the latter is destroyed, is absent when the subject is in energy balance, and therefore is of no significance here. Finally, the most interesting fact is that the "water as such," or the free fluid of the intake, constitutes less than 50 per cent of the total water available to the subject. Dederding determined only the free fluid intake in her investigations.

In considering the various losses of water from the body, a most surprising amount is lost by vaporization from the skin and lungs. This loss is comparable to the amount of water lost as urine and is rarely determined by those pretending to measure water exchange. The water contained in the stool is relatively small, but it is apparent that it, too, must be determined in an accurate water exchange. The water of the urine is the one loss most commonly

determined, but it constitutes less than 50 per cent of the loss of water from the body. The quantity lost by vaporization is often greater, although it has seldom been considered in studies of water exchange. Dederding, in her investigations, determined only the urine volume.

It must be obvious that determination of the free fluid of the intake and the urine volume constitute less than 50 per cent of the total exchange of water by the body. Consideration of only these two items of water exchange often result in erroneous conclusions regarding the status of body water. The following examples (Tables 2 and 3) are given to demonstrate this fact. Table 2 shows the data obtained from a postoperative case of acute intestinal obstruction.

TABLE II.—WATER EXCHANGE—DEHYDRATION.

AVAILABLE WATER.	Grams.	EXCRETED WATER.	Grams.
1. Exogenous		3. Urine	700
A. Diet	0	4. Stool	750
B. Water as such	3000	5. Vapor	1000
2. Endogenous		6. Emesis	1500
A. Oxidation	225	7. Drainage	750
B. Preformed	200		
Total	3425	Total	4700
Daily dehydration 1275.			

If one compared only the "water as such" (3,000 grams) with the urine water (700 grams), the daily dehydration of 1,275 grams would never have been suspected. Table 3 shows the data obtained from a subject having chronic nephritis and edema. If, in this subject one compared the "water as such" (1,200 grams, which is smaller

TABLE III.—WATER EXCHANGE—HYDRATION.

AVAILABLE WATER.	Grams.	EXCRETED WATER.	Grams.
1. Exogenous		3. Urine	800
A. Diet	900	4. Stool	150
B. Water as such	1200	5. Vapor	850
2. Endogenous			
A. Oxidation	250	Total	1800
B. Preformed	0	Daily hydration 550.	
Total	2350		

than that of the subject in Table 2) with the urine water (800 grams, which is larger than that of the subject in Table 2), the daily accumulation of 550 grams of edema would have been overlooked.

The foregoing facts make it evident that investigations similar to those of Dederding are of value only when a method which insures a more accurate determination of water exchange is employed. The data presented below is the result of such an investigation.

Time does not permit a detailed account of the procedure by which an accurate statement of water exchange was obtained in this investigation. For these details, reference may be made to the original descriptive articles.[1] However, the following brief description is given in order to emphasize the care and precision which are imperative in such a study.

The subject was weighed in the nude state at definite intervals of either twelve or twenty-four hours on a special balance which is accurate to 1.0 gram. The drinking water ingested daily was accurately weighed in a large thermos bottle from which the patient drank through a glass tube. Throughout the experiment, the diet was constant, the same kinds and amounts of food being ingested day after day.* The food was prepared, accurately weighed and even served by one of us. The water content of the food was determined by dessication. The urine and stool were collected daily in closed pails, accurately weighed and the water content of each also determined by dessication. The daily excretion of nitrogen was obtained by Kjeldhal analysis of the urine and feces. The water lost by vaporization from the skin and lungs, and that available from the oxidation of the foodstuffs, was obtained by means of the insensible loss of weight and the metabolic mixture as described by Newburgh, Wiley and Lashmet.[2]

Thus, in this investigation, the water exchange was determined by quantitatively measuring every item of water "into" and "out of" the body.

The data presented in Fig. 1, was obtained from a patient who had had typical Ménière's disease for ten years. For about two years prior to this study he had averaged two or three attacks per week, and for the past six months had suffered a seizure daily. These attacks were characterized by a "feeling of fullness" in the head, "buzzing and ringing" in the ears, dizziness, and finally, nausea with vomiting. The only relief he had been able to obtain was secured by lying very quietly with eyes blindfolded.

*The daily diet consisted of: Milk, 600 gms.; cream (40%), 100 gms.; cheese, 100 gms.; bread (whole wheat), 150 gms.; puffed wheat, 10 gms.; grape fruit (canned), 100 gms.; butter, 60 gms.; apple sauce, 150 gms.; sugar, 10 gms. Such a diet contained: Nitrogen 9.7 gms.; fat, 154 gms.; carbohydrate, 188 gms.; calories, 2382; total solids, 397 gms.; sodium, 87 milli-equivalents.

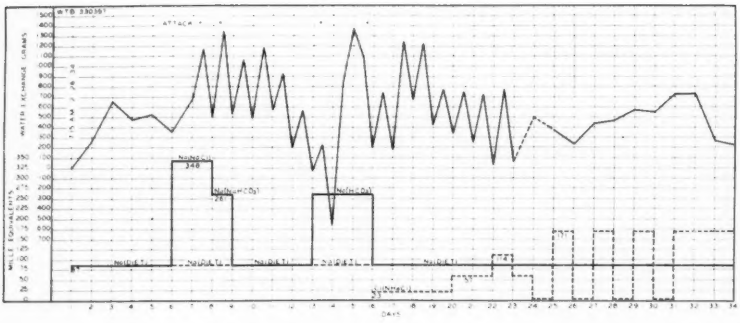


Fig. 1.

For the first six days of this investigation, water exchange was determined for periods of twenty-four hours. It was soon realized that changes in body water occurred so quickly that twelve-hour periods of determination were necessary. Consequently, for the remainder of the study, water exchange was measured for periods of twelve hours, one period beginning at 7:15 A. M. and ending at 7:15 P. M., and the following period beginning at 7:15 P. M. and ending at 7:15 A. M.

The first five days were used as a control period, in an attempt to estimate the status of body water. The subject received the constant diet, as already described, and was permitted to drink water freely. During the first two days there was considerable retention of body water, half of which was lost spontaneously in the following three days. There were no attacks during this period.

On days 6 and 7 the subject received 348 milli-equivalents of sodium (87 m.-eq. in the diet and 261 m.-eq. as sodium chlorid), administered daily, during the twelve hours while awake. For the first thirty-six hours there was a marked retention of water in the body. A very severe attack, as previously described, occurred at the height of hydration. During the following twelve hours there was a spontaneous loss of water with a distinct diminution in the severity of the symptoms. During the first twelve hours of day 8, the subject ingested 261 milli-equivalents of sodium (87 m.-eq. in the diet and 174 m.-eq. as sodium bicarbonate). Again, there was a marked hydration with a second attack occurring at its height. During the following twelve hours, all of the water which had been held during the preceding twelve hours was released and the subject again improved. It will be noted that these two attacks occurred

when sodium was administered and retention of water by the body was in evidence. Consequently, one cannot assume that either one of these factors is solely responsible for the attacks.

In order to study separately the effect of sodium administration and retention of body water, the subject was purposely dehydrated during days 11, 12 and 13, by rigidly restricting the intake of water. When the subject was dehydrated to a level at which he had previously had no attacks (control period) he was then given 261 milli-equivalents of sodium (87 m.-eq. in the diet and 171 m.-eq. as sodium bicarbonate), during the first twelve hours of day 13. A severe attack occurred, in spite of the fact that the body contained strikingly less water than it did when the attacks were absent (days 1 to 6 and 9 to 11). During the first twelve hours of day 14, the same intake of sodium was maintained and water was freely given. There was marked retention of body water, although no attack was produced. However, the following day (day 15), during which the same intake of sodium was maintained, a fourth attack occurred, but not until the subject had lost approximately 500 grams of the water previously stored.

Beginning with day 16 and continuing to day 34, the intake of sodium was only that contained in the diet (87 m.-eq.). Water was permitted and averaged about 1,800 grams daily. Ammonium chlorid, an acid-producing salt, was administered in increasing dosage. At first, water was retained but this was gradually lost. During the period of water retention, it will be noted that the amount of water held was practically as great as when sodium was administered, but attacks resulted when sodium was given, while none were produced by ammonium chlorid. Since ammonium chlorid administration is associated with an increased excretion of sodium, it is presumed that ammonium chlorid failed to produce an attack because it prevented the retention of sodium by the body.

It is obvious from this data that attacks occurred quite independently of whether water was stored or lost by the body. Furthermore, attacks were produced by the administration of sodium, regardless of whether such administration was attended by hydration or dehydration. The acid-producing salt, ammonium chlorid, did not produce an attack, in spite of the fact that it was at first attended by a retention of body water quite as great as that accompanying ingestion of sodium.

SUMMARY.

It is assumed from our experiments that the symptoms of Ménière's disease are due to the retention of sodium by the body. If

a sufficient supply of water is available it will likewise be retained, but this fact is not of primary importance. Apparently, the local tissues involved in Ménière's disease have either an increased avidity for sodium or an unusual sensitivity to it.

The therapeutic indications are: (1) to permit as small an intake of sodium as is possible and (2) to prevent the accumulation of sodium by the body. The former is easily attained by means of diet and the latter by the administration of acid-producing salts, such as ammonium chlorid. When these two factors have been controlled, the intake of water does not need to be considered.

We have treated fourteen cases of typical Ménière's disease successfully by this method. Each patient was carefully selected in an effort to rule out any obvious lesion, such as an acoustic nerve tumor or a suppurative process in the temporal bone, and every precaution was taken to eliminate psychical elements from this study.

While the number of patients is not large, it represents a group carefully chosen from the cases available in our clinic during the past ten months. Each patient was hospitalized for a period of thirty days or more and the efforts of two members of the staff and myself were directed daily towards a careful clinical and laboratory study of the problems at hand.

It is of extraordinary interest to note that identical results were obtained for all of the patients in this group. In not one instance did we fail to produce an attack by the administration of sodium, and not once were we disappointed in obtaining complete relief by the medical therapy above described. The patient whose chart is herein presented has been without a seizure since ammonium chlorid was first given to him on October 16, 1933, and similar results for shorter periods have been obtained for the remaining thirteen cases in the group.

We are fully aware that presumptuous claims for medical therapy cannot be based upon the meager statistics herein compiled. It is not our wish that such an inference should be drawn. Rather do we desire to suggest a new etiologic factor in the production of Ménière's disease and express the hope that it may lead to further research in therapeutics.

Obviously a study of this kind leads to many ramifications in the field of clinical medicine and physic and biochemistry. With each original discovery, new problems for further investigation are brought to light. Innumerable questions promptly arise and critical suggestions are prone to appear. Both are invited. We ask for criti-

cism with the hope that it will criticise the work which is presented rather than the problems which the study suggests.

TREATMENT.

1. Protein, unrestricted or forced.

2. Calories, as indicated.

3. Low salt content.

4. Medication. Ammonium chlorid, 3.0 gms., with each meal, in capsules (six capsules each containing 7½ grains taken during the meal), three days on and two days off. The capsules should not be replaced by the chocolate coated or the enteric coated pills because the latter sometimes pass through the gastro-intestinal tract without being absorbed. The ammonium chlorid can be given in this dosage for an indefinite time without injurious effects. We have had patients with nephritis receiving ammonium chlorid in this manner for a period of five years.

5. Water intake unrestricted, although excessive quantities of liquids should not be taken.

6. Diet, approximate neutral, low sodium diet.

Group A: The following foods may be taken daily:

1. Eggs, meat, fish and fowl as desired.
2. Bread as desired.
3. Cereal, one of the following: Farina, oatmeal, rice, puffed rice or puffed wheat.
4. Potato and one or more servings of any of the following: (a) macaroni, (b) spaghetti, (c) rice, (d) corn, (e) cranberries, (f) prunes, (g) plums.
5. Milk as desired.
6. Vegetables and fruits daily of any fruit and of any vegetable not included in groups "B" and "C" as desired.
7. Butter, cream, honey, jellies, jams, sugar and candy permitted as desired.
8. Tea and coffee as desired.

FOODS TO BE AVOIDED.

Group B: All salt meats and fish, or bread, crackers and butter prepared with salt. Carrots, clams, condensed milk, raisins, caviar, cowpeas, olives, spinach, cheese, endive, oysters.

FOODS TO BE TAKEN NO MORE THAN TWICE WEEKLY.

Group C: Lima beans, beets, buttermilk, cantaloupe, cauliflower, celery, chard, dried cocoanut, dried currants, dates, figs, horseradish, kohlrabi, limes, muskmelon, peanuts, peaches, mustard, pumpkin, radishes, rutabagas, strawberries, turnips, turnip tops, watercress.

NOTE: All food to be prepared and served without salt.

BIBLIOGRAPHY.

1. Dederding, Dida: Acta Otolaryngologica, 1929, Sup X, Vol. 1; also 1931, 16:404.

2. Newburgh, L. H.; Wiley, F. H., and Lashmet, F. H.: Journ. Clin. Invest., 1931, 10:703; also 1931, 10:723.

LXXXV.

CARCINOMA OF THE TONSIL: A STATISTICAL STUDY OF 230 CASES.*

LE ROY A. SCHALL, M. D.,

BOSTON.

MATERIAL.

From 1918 to 1934, 230 patients with carcinoma of the tonsil were seen by the laryngologic staff of the Collis P. Huntington Memorial Hospital. This paper is a statistical study of these records in an attempt to find some ray of hope in the treatment of this disease. In the same period of time, 24,437 patients with malignant diseases were admitted to the hospital, so that the incidence of tonsillar involvement is almost one in every one hundred and six cancer patients.

Year	Cases
1918	14
1919	16
1920	9
1921	26
1922	21
1923	10
1924	17
1925	16
1926	13
1927	16
1928	16
1929	11
1930	14
1931	9
1932	11
1933	11
	230

The yearly incidence runs very evenly—the greatest number, 26, were seen in 1921; the fewest, 9, each in 1920 and 1931; so that, apparently, in as far as this series is concerned, there is no evidence of an increase in the incidence of the disease.

*Read before the annual meeting of the American Laryngological Association, Cleveland, Ohio, June 8, 1934.

NATIVITY.

American	108
Canadian	22
Irish	35
English or Scotch	15
Scandinavian	3
German	4
Russian or Baltic	3
South or East Europe	11
Unknown	27

In the racial study of this disease, the incidence of occurrence is in direct proportion to the racial groups served by the hospital. It is natural that at this hospital the greatest group is found in those of American nativity, with those of Irish and Canadian ancestry forming the second and third groups in order of frequency.

OCCUPATION.

Farmer or Laborer	52
Skilled laborer	63
Factory worker	19
Clerk	8
Professional	11
Commercial	13
Transportation	10
Housewife	18
Domestic	6
None	7
Unknown	7
Other	16

It is natural also that the greatest groups should fall into the greatest social groups, i. e., the laborer and the skilled labor classes.

SEX.

Male	203—88.7 per cent
Female	27—11.3 per cent

The incidence in the male sex is most striking—203 males, or 88.7 per cent, and 27 females, or 11.3 per cent.

MARITAL STATUS.

Single	36
Married	139
Widowed	45
Divorced	5
Unknown	5

AGE.

30-40	4
40-50	19
50-60	72
60-70	86
70-80	38
80 and over	8
No record	3

The age group is that of past middle life. In the more detailed study, the greatest incidence is found in the group between 60 and 65 years of age; although two were below 35, and eight over 80 years of age.

WEIGHT.

In studying the weight charts of these patients, it is found that there is no one group that predominates. From 96 to 220 pounds—the incidence of involvement differs but little.

LOSS OF WEIGHT.

Fifty-three of these patients showed no loss of weight; 33 lost less than 10 pounds; 11, up to 20 pounds; 7, up to 40 pounds, and one more than 40 pounds in weight. Sixty-five stated they had lost weight but were uncertain of the amount. Contrary to all expectations, five had gained weight, while 55 records made no mention of the weight of the patient.

FAMILY HISTORY.

In only 18 of these 230 patients was there a family history of cancer. Only 8 gave a history of syphilis, while only 7 had positive serologic tests. Forty-two patients were nonusers of tobacco.

TOBACCO.

None	42
Pipe	27
Cigar	11
Cigarettes	7
Smokes (method not stated)	136
Chews	45
Unknown	12

In only 48 of these 230 cases was the disease limited to the tonsil. I have made no attempt to determine the site of the primary lesion. In addition to the tonsil:

The palate was involved in	76, or 33.	per cent
The pyriform sinus in	50, or 21.7	per cent
The tongue	48, or 20.8	per cent
The anterior pillar	34, or 14.7	per cent
The postpharyngeal wall	18, or 7.8	per cent
The posterior pillar	18, or 7.8	per cent
The lower jaw	8, or 1.3	per cent
The upper jaw	8, or 3.4	per cent

TYPE OF LESION.

Examination showed the lesion to be ulcerated in 98; tumor in 132; to involve the right side in 127; the left in 103.

SYMPTOMS.

The study of the patient's first symptoms shows that 104 had pain, 84 first complained of growth, 4 showed bleeding, 9 had hoarseness, 4 ulceration, 14 dysphagia, 10 dysphonia and only 1 leucoplakia.

DURATION OF SYMPTOMS.

Less than two weeks	4
Fifteen to thirty days	14
Thirty-one to ninety days	65
Three to six months	70
Six to nine months	22
Nine to twelve months	28
Thirteen months to two years	10
Over two years	10
Unknown (no record)	7

In a region as easily examined under direct vision as the throat, it is surprising that these patients endured their symptoms on an average of three to six months before consulting a physician. Only four sought advice in less than two weeks. Many waited until enormous cervical metastasis occurred. In the over two-year group these had undergone previous treatment elsewhere and came for recurrence.

PREVIOUS STUDY.

None	101
Yes	129

Although 129 were referred to the hospital by a physician, only 44 received prior treatment. An analysis of this treatment showed that there was a local operation on the growth in 27 cases; radical neck dissection on 2; incomplete neck dissection on 4; radium was used on 15 and deep x-ray radiation on 17. Five were treated for syphilis.

GLANDS.

Glandular enlargement occurred fairly early. Only 19 showed no evidence of glands. Their distribution showed enlargement: on

the right, 75; left, 69; both sides, 22. Submental, 2; submaxillary, 45. The glandular enlargement varied from small palpable glands to tremendous masses in the neck, of which there were 41 cases.

PRETREATMENT COMPLICATIONS.

None	167
Old age	34
Heart, arteriosclerosis	13
Nephritis	1
Diabetes	4
Malnutrition	4
Anemeia	1
Other disease	6

PATHOLOGY OF TUMOR.

Epidermoid carcinoma, Grade I	38	24.09	per cent
Epidermoid carcinoma, Grade II	57	37.	per cent
Epidermoid carcinoma, Grade III	52	33.7	per cent
Transitional cell carcinoma	7	4.5	per cent
Ungraded	76		

As this study included cases prior to Broder's[1] classic classification of malignancy, 76 cases are listed as ungraded.

TREATMENT.

Since 1918 the treatment of malignant disease of the tonsil has passed through three distinct phases. The first phase may be said to be that of surgery. The surgical procedures varied from simple enucleation of the tonsil to that of ligation of the carotid, radical neck dissection and a wide radical dissection of the growth. With the advent of electrosurgery, diathermia and electrocoagulation has replaced the scalpel.

During the past three years at this hospital, the treatment consists of the combined use of radium and high voltage roentgen ray therapy. Radium is used for the treatment of the local lesions and x-ray for the external radiation of the neck. The radium treatment always consists of radon implants in the form of gold seeds, having the wall thickness of .2 mm. offering a filter sufficient to remove practically all beta radiation. These seeds are dispersed throughout the local lesion, an effort being made to place them in such a way that the lesion will receive uniform irradiation. By placing the seeds as nearly as possible 1 cm. apart, all portions of the lesion will receive a radiation of nearly uniform density, which can be varied to suit the case by using seeds of different contents of radon.

As a rule, radon seeds of 1 mc. strength are employed. This, however, should not be made a strict rule. The total amount of radon

used has varied from 2 to 12 mc., according to the size of the lesion. From time to time definite recommendations as to the dosage have been made in various clinics. In this group of cases a rigid rule has not been followed; particularly in cases with large lesions the dosages have been very much less than those recommended. In the use of radium we should always remember that a caustic effect is produced, not because the caustic rays have not been filtered, but because the biologic effect in the immediate vicinity of the radium implant is so great that normal tissue and tumor tissue are destroyed. The extent of the slough depends on the strength of the radium seed. The same is true in the case of other forms of radium implants, if too large doses are used in too small areas.

The external radiation is given through the use of short wave roentgen ray produced at 190 kilovolts constant potential, and filtered through copper of ½ mm. thickness. The skin focal distance is 45 cm. It is felt that the neck can be irradiated satisfactorily in this manner, and much more easily and effectively than with the use of external radium pack. The use of radium bomb, such as advocated in various clinics where a large amount of radium is available, has not been tried here, because of the insufficient amount of radium.

Only one side of the neck, the affected side, is exposed, unless it is felt that the disease has extended to the other side of the neck. The doses range from 2000 to 4000 r units. The international r unit is used, and the measurements given are those taken in air. In this way a mild erythema can be produced by giving 800 r units at one sitting. The total dose is given in fractional amounts of 300 to 600 r units over a period of from five to forty days. A dose of 2000 r units given in 400 r units amounts on successive days is perhaps the least from which we can expect results.

Commonly we have given an initial dose of 2000 r units, then allowed a period of two to three weeks to elapse to judge the amount of regression in the tumor, if any, and also the local and general reaction. If the response to treatment in the growth has been sufficient and the condition of the patient has remained good, the treatment has been extended and as much as 4000 r units have been given in forty days without producing loss of more than the superficial layers of the skin.

External radiation is usually carried out in the week following the insertion of the radium implants. Occasionally where the primary tumors have been of very large size, external radiation has been given first in an attempt to produce some diminution in the size of the tumor and permit more effective use of local treatment by radium.

SUMMARY OF TREATMENT.

No treatment	13
Treatment, not completed	99
Operation and radium	10
Operation and x-ray	4
Radium and x-ray	104

Thirteen patients received no treatment. They came either for diagnosis or confirmation of the diagnosis. Ninety-nine did not complete their treatment. These were the patients that were hopeless when first seen. Ten were treated by operation followed by radium, while four received deep x-ray therapy following operation. One hundred and four have been treated by radium implantation followed by deep x-ray. The results of this study are based, therefore, on 118 treated cases.

RESULTS.

No relief	43	39.3	per cent
Relieved, no recurrence	23	18.4	per cent
Recurred under 1 year	47	39.8	per cent
Recurred in 1-2 years	2	1.01	per cent
Recurred in 2-5 years	2	1.01	per cent
Recurred after 5 years	1	.5	per cent

Of the twenty-three patients who have had no recurrence, five have died a noncancerous death.

The end results of all cases as of January, 1934, show that there are:

Dead	202
Lost	6
Living	22

Of the 22 patients now living, 18 are without clinical evidence of cancer.

Year	Number of Cases	Number Healed	Percent	Duration
1921	26	1	3.8%	12 years
1924	17	1	5.8%	9 "
1925	16	1	6.2%	8 "
1928	16	1	6.2%	5 "
1929	11	1	9. %	4 "
1931	9	3	33.1/3%	2 "
1932	11	5	45.4%	1 "
1933	11	5	45.4%	Less than 1 year

From a statistical point of view, these percentages compare favorably with those of Berven[2] and Coutard.[3]

Clinically the results of treatment are most encouraging, even though a "cure" may not always be obtained.

It is our experience that this method of treatment produces the quickest results without incapacitating the patient or subjecting him to a long hospitalization.

CONCLUSIONS.

1. In certain selected cases, surgery may still have a place in treatment of malignant disease of the tonsil.

2. When the lesion has spread beyond the tonsil, the combined use of radon implantation and external irradiation has given us our most encouraging results.

I wish to express my indebtedness to Dr. Charles Dumas for his technical aid in describing "Treatment."

270 COMMONWEALTH AVE.

REFERENCES.

1. Broders, A. C.: "Squamous Cell Epithelioma of the Lip." J. A. M. A., 74, 656, 1920.

2. Berven, E.: "Malignant Tumors of Tonsils." Acta Radiologica, Supp. Xi (1931).

3. Coutard, H.: "Roentgenotherapie des Epitheliomas de la region amygdalienne." Radiophys. et Radiotherapie, 1930-1932 (ii), 541 et seq.

LXXXVI.

THE APPLICATION OF VIABLE MUSCLE IN VASCULAR INJURIES.*

O. Jason Dixon, M. D.,

Kansas City, Mo.

The application of viable muscle for the control of bleeding in vascular injuries actually dates beyond the beginning of man, and had it not been for certain physiologic reactions that always take place following the damage to the blood vessel we would not be here today to discuss this subject. At present, however, we are just beginning to understand a few of the physiologic and pathologic principles that have long been applied by nature in the control of this process. The entire problem hinges upon the study of thrombosis, the purpose of which we understand much more clearly than the manner in which it is performed. A tremendous amount of work has been done upon thrombosis, and the wide divergence of opinion which exists clearly demonstrates that it is not yet thoroughly understood.

Mills[1] in his investigation on the physiology of blood coagulation has described a very important substance which he calls "tissue fibrinogen." This tissue fibrinogen has an enormous coagulant action and is found in the plasma of every cell in our body. Different tissues, however, possess different amounts of this substance and, as vascular endothelium is very rich in it, it is quite obvious that the blood which first comes in contact with the injured endothelium is greatly affected by this primary injury. Certain other tissues contain large amounts of tissue fibrinogen—the lungs, brain, kidneys, heart and skin in the order named. Mills identified tissue fibrinogen as the globulin fraction of tissue extracts; the albumins possessing no action of any kind in the clotting process. If injected intravenously with sufficient rapidity, intravascular coagulation takes place so rapidly that death occurs within one minute. The blood platelets also contain different very important constituents of coagulation and at times may become an important factor in the formation of the thrombus. Howell[2] has identified an active agent of tissue juices and platelets as "cephalin." Morris[3] has shown that scrapings of the intima of blood vessels is

*From the Department of Otology and Experimental Pharmacology, University of Kansas School of Medicine.

more active in the formation of blood coagulation than lung extracts. Although there is a difference in opinion, it is probable that the properties within the blood as well as the vascular and extravascular tissue juices all combine to hasten and complete the normal physiologic process of thrombosis. Time does not permit the exposition of these very interesting and instructive phases of thrombosis. Briefly, thrombosis may be accepted as a clotting of blood in living vessels which is a block formation arising from the constituents of the circulating blood and leading to complete closure of the vessel lumen. Its functions may be defined first, as the prevention of hemorrhage in a damaged vessel wall; second, the covering of bacteria and the localization of their action; third, as an attempt to prevent an erosion or invasion into the blood stream. This may be either infectious or traumatic in origin.

CLINICAL APPLICATION.

The major clinical application in the studies of this problem have been directed to thrombophlebitis of the sigmoid sinus in relation to mastoid disease. At times it may happen that I have digressed far from the field, but regardless of the location of the lesion, the pathologic and physiologic changes which occur in vascular injuries are almost identically the same. In my experimental studies upon the dog, principally on account of the anatomic differences to the structure in the human, I have been obliged to depart from areas that would be directly comparable to those in the human and have applied this investigation to such remote venous channels as the inferior vena cava, the femoral vein and the abdominal viscera, such as the liver, spleen and kidney. The mastoid process and adjacent intracranial sinuses in the dog are in no way comparable to the human. The brain is much smaller in comparison and therefore uses much less blood. Even the veins in the neck of the dog are reversed in their proportionate size. The external jugular vein being much the larger in this animal and, as it is quite superficial and very accessible, was used for the major portion of these experimental studies. As the dog's principal blood supply of the head and neck goes to the muscles of mastication and to the salivary glands, the latter playing a very important part in the maintenance of the temperature equilibrium, it readily explains these vascular departures from the human.

EXPERIMENTAL PROCEDURES.

The first studies that were made were to determine what physiologic changes took place when the external and internal jugular veins were completely obstructed. Compensatory collateral circulation

principally through the subcutaneous veins develops so quickly that no disturbance to the animal whatever could be discerned. Experiments were then conducted purely as a repetition of the old established procedures as have long been used in the human for the control of bleeding namely, ligation of the vessel, damage to the intima carried out by curetting the lining of the vessel wall proximal to the ligation and the insertion of foreign plugs directly into the lumen of the vessel, such as cotton, rolled gauze and fine mesh cotton fabric. The reactions in these experiments were always the same. Bleeding was controlled by the formation of the thrombus within the vessel, and except in a few cases where the plug was spontaneously expelled too soon there was no postoperative hemorrhage.

In comparison to the use of viable muscle for the control of bleeding, which I shall discuss later, there were several things which happened in the use of these foreign bodies which did not compare favorably with viable muscle. First, the immediate control of bleeding was delayed, particularly if the plug did not completely occlude the vessel lumen. Unless the plug was compact there was leakage through it. The extent of the retrograde thrombus was only one-fourth to one-sixth as long as that of the viable muscle. The clot was not firm and was not as intimately attached to the vessel wall. Necrosis of the vessel about the site of the foreign body usually took place, and the plug was spontaneously expelled from the seventh to the twenty-first postoperative day. This was not, however, followed by hemorrhage as shown by another very important finding, namely, that the vein, which was obstructed by these plugs, never recanalized past the point of obstruction. Infection occurred much more frequently and played a part in the spontaneous expulsion of the plug. When these plugs were removed too soon, i. e., twenty-four hours or thirty-six hours postoperative, the thrombus which had formed was so adherent that it came away with the plug and bleeding recurred. In a few instances the plug was retained and encapsulated as a sterile foreign body. (Fig. 1.) These experiments clearly demonstrate the disadvantages of gauze for the control of hemorrhage. Similar experiments upon the external jugular vein were then conducted with the use of nonviable muscle. Nonviable muscle was not nearly as satisfactory as the use of gauze plugs when inserted directly into the lumen of the vessel. The principal reason is that they become loosened and are more easily expelled. In two animals that had stormy convalescences from their anesthesia there were fatal hemorrhages and the muscle plugs were spontaneously expelled. In three animals it was necessary to ligate the bleeding vessel. Infection

Fig. 1. Cross section of external jugular vein with gauze implant. Thickened vein wall and no attempt at recanalization past the foreign body obstruction.

was more frequent about this nonviable muscle plug. The thrombus which occurred was, however, more extensive than with the gauze plugs. The reactions in the vessel wall (Fig. 1) which took place at the site of the nonviable muscle implant was so extensive that recanalization did not take place.

The next series of experiments were somewhat similar to the work which was originated by Sir Victor Horseley,[4] namely, the use of muscle tissue taken directly from the animal's own body. It is interesting to digress a moment and recall that Horseley was the first man to use bone wax for the control of bleeding, a method which he states he obtained on a hint from a Magendie's Physiology. After this he used a cut fragment of muscle from the injured surface of the wound and soon learned that the muscle fragment closely adhered to the wounded tissue to which it was applied. He carried out experiments upon the brain, the liver and even the divided arteries,

including the aorta. He then boiled this tissue and found that it had a very poor hemostatic effect. Fascia was tried and proved unsatisfactory. He says that he has used it freely in operations; above all, when it was necessary and convenient to leave a resting plug or tampon in a wound, and he ocasionally had the opportunity to use the isolated muscle tissue. He found it firmly attached and organized and in no instance did any ill effects follow its employment.

VIABLE MUSCLE.

So far as I have been able to determine, viable muscle for the control of hemorrhage and the repair of vascular injuries has not been tried. Brain surgeons have, however, for many years applied with great success the use of nonviable muscle segments, as originally described by Horseley. It is the application and the results of viable muscle with the control of bleeding and the part which it plays in vascular repair that I especially wish to report. The unsatisfactory experiences which I had in the management of postoperative sigmoid sinus thrombosis prompted my researches in this field. I have searched diligently for several years for some substance that would promptly and thoroughly control the bleeding from the opened sigmoid after the infected thrombus was removed and yet would not necessitate the postoperative disturbance of the injured vessel or the newly formed thrombus. Frequent unpleasant experiences taught me the dangers that might arise from tampering with this infected vessel. While the gauze plug which has been so universally used since the first operation upon the sigmoid sinus, very satisfactorily controls the bleeding, yet that is only a minor part in the successful completion of the operation. This plug is a foreign body which is inserted into an infected field. There is always a certain amount of pressure necrosis which is distinctly undesirable when adjacent to the dura. But the most perplexing problem to me has been the removal of this plug. If removed too soon the newly formed adherent thrombus comes away with the plug and the dangerous process of repacking must be repeated. If left too long, the foreign body reaction promotes infection of the newly formed adjacent thrombosis. In an attempt to escape from this midway and most uncomfortable position, I have used viable muscle which, fortunately, is always at hand, easily obtained, and without any particular discomfort to the patient and no untoward risk or harmful after effects. This viable muscle is the sterno-cleido-mastoideus, which is obtained merely by lengthening the lower angle of the mastoid incision (Fig. 2), lowering the bony tip of the mastoid process, dividing the muscle longitudinally for a desirable size and sectioning this divided portion six to seven centi-

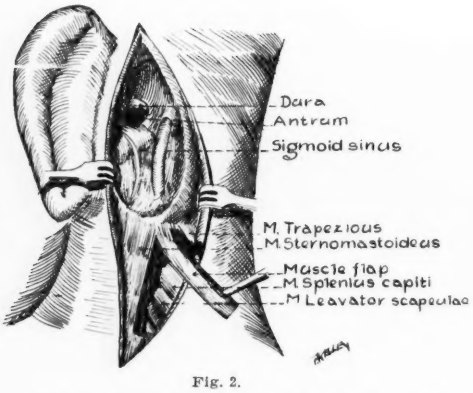


Fig. 2.

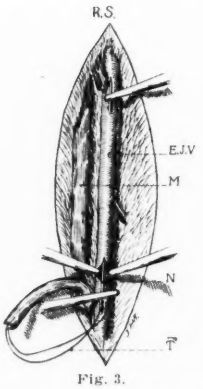


Fig. 3.

Fig. 2. Opened mastoid wound with the viable muscle strip ready to be placed over the opened sigmoid sinus.

Fig. 3. Needle leading the viable muscle into the opened vein. (Dog 32, Operation No. 1, March 17, 1933.)

meters below its attachment to the mastoid tip. The muscle flap is then turned upward and, by finger pressure only, is held over the sigmoid sinus wound. This quickly and firmly adheres to the vessel wall and the tissue juices of the raw muscle hasten the formation of a firm thrombus. The sigmoid sinus need never again be disturbed.

Quite obviously, I have again been obliged to turn to the dog in order to determine what happens physiologically, pathologically and anatomically after this application. I have attempted to discover all the disadvantages that might arise, and I am happy to state that these have been greatly diminished in my mind by the results of my experimental researches and, much to my gratification, I have discovered several unexpected advantages. The anticipated disadvantages were as follows: First, the failure of the viable muscle to safely and completely control bleeding; second, infection with necrosis and sloughing of the viable muscle flap; third, the extension of the infection into the neck wound at the site of muscle section. None of these complications have arisen and from my experimental work I am led to believe that they are more imaginary than real. I have learned, however, to avoid trauma as much as possible to the viable muscle strip and that it should be kept warm and moist and should not be twisted or drawn over an obstructive point and should not be left inserted under any tension. Due to the rapid escape of tissue juices and the formation of the clot at the site of the injured muscle there are advantages in applying the raw surface of the muscle to the injured vessel as soon after the muscle is sectioned as possible. The muscle may be kept at

body heat and moist by leaving it in its own bed until ready for use. It is better to make one application and not remove it. It is also an advantage to have the raw surface of the muscle approximate the raw edges of the vessel. Blood clots between the two apposing surfaces should be avoided as much as possible. In handling it, instruments should be applied only at the cut end, as this area is already traumatized by the incision and it does not interfere so much with the blood supply to the tissues. Blunt dissection should be used as much as possible and lines of cleavage in the muscle should be followed in the dissection. The normal blood and nerve supply to this muscle should be disturbed as little as possible. While I am satisfied that the application of this viable muscle to the opening in the sigmoid will safely and completely close the wound in the vessel and prevent the recurrence of hemorrhage, yet additional safety may be secured by leading the muscle into the lumen of the vessel with a curved needle and thread tied to the end of the muscle. It is led into the vein in the same manner that the viable muscle was led into the external jugular vein of the dog. (Fig. 3.) The sides of the muscle can also be tacked to the edges of the opened sigmoid sinus. Precautions should be taken in order not to pass the needle through the dura. This procedure will temporarily fix the muscle into position, and as a thrombus forms so quickly after this application there is very little danger of postoperative hemorrhage.

POSTOPERATIVE REPAIR OF THE OPEN SIGMOID SINUS.

I have never had the opportunity to study the sigmoid sinus and determine just what reparative processes took place after it has been opened and packed with gauze and the patient has completely recovered from the operation. From my experiments upon the dog, I do not believe that the vessel recanalizes when gauze plugs are used. I do know that in the dog, when viable muscle implants or explants are used, that the vein always recanalizes past the point of the venous obstruction caused by the viable muscle plug. Recanalization (Fig. 4a and 4b) begins as early as the seventh postoperative day and the vein rapidly enlarges past this point of obstruction so that within thirty or forty days it has returned to its normal size and capacity as a blood carrier.

In one patient, upon whom I have used a viable muscle explant to repair the open sigmoid sinus, I am firmly convinced, only by external inspection during the healing of the mastoid wound and after it had completely healed, that the sigmoid is patent. I have been able to demonstrate by compression on the corresponding internal jugular vein a bluish distention that can be seen and palpated beneath

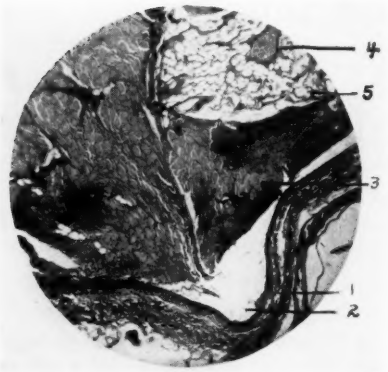


Fig. 4a. Cross section through external jugular vein with viable muscle implant fourteen days after insertion. *1* vein wall, *2* newly formed channel, *3* cross section of viable muscle fibers, *4* nerve, *5* fat.

the thin mastoid scar. Of course, this observation is not conclusive evidence of recanalization and could only be completely confirmed by operative or necropsy studies and also by the injection of opaque media into the upper end of the sigmoid sinus for the use of an x-ray picture. I would like very much to make x-ray studies in this case, but of course do not feel justified in carrying out this procedure.*

We have known for some time that the infected and thrombosed sigmoid sinus that is not operated upon may recanalize. Recently I have seen such a specimen that was removed from a child that had died from a complicating meningitis, three weeks following the onset of the thrombophlebitis. I also have observed several patients who made a spontaneous recovery from the thrombophlebitis of the sigmoid sinus when the sigmoid was not opened. When there is bilateral sigmoid sinus involvement or where the wrong sigmoid sinus is opened at operation it may be a distinct advantage to the patient for recanalization and restoration of the normal function of the sigmoid sinus as a blood carrier to occur.

In order to study the intravascular reactions in these experimental studies upon the dog without opening into the vessel, I have

*Since this paper was read, two injections of opaque media were made directly through the skin into this sigmoid sinus, which showed conclusively that the vessel was patent (September 28, 1934). I have also made nineteen injections into the longitudinal sinus and exposed sigmoid sinus on the living patients with various concentrations of two types of inert iodin preparations in glucose that have been chilled. Very definite valuable information was obtained in all the cases where proper solution was used without any disturbance to any of the patients. Details of this method and types of solution used will be presented in a separate paper.

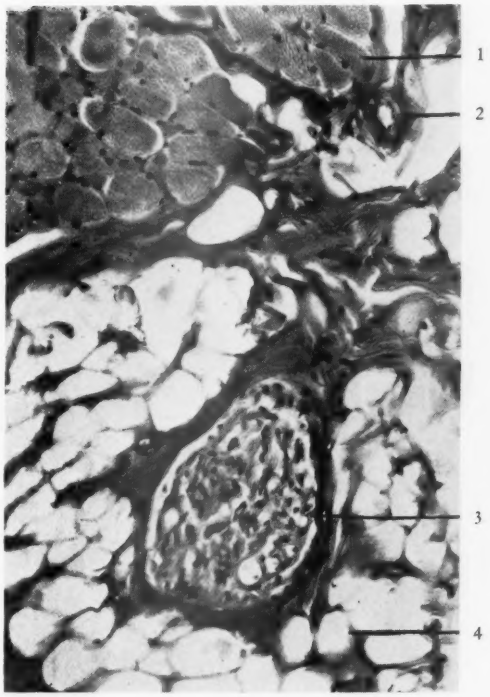


Fig. 4b. Part of the cross section of viable muscle implant fourteen days following insertion into external jugular vein. *1* cross section of viable muscle fibers. *2* newly formed blood vessel within the muscle, *3* cross section of nerve carried in with the muscle, *4* fat cells.

used a concentrated solution of neo-skiodan and 50 per cent glucose injected into the sublingual vein and have taken the x-ray picture while the opaque media was being injected. This has enabled me to determine the extent of the primary thrombus, the beginning of the recanalization and the restoration of the venous channel past the point of obstruction. (Fig. 5.) Unfortunately, I always found it necessary on account of the rapid dilution in the blood stream to inject it distal to the point of the venous obstruction. In using this method for radiographic studies on the sigmoid sinus it would obviously be necessary to inject the media directly into the dural sinus above the point of venous obstruction. This entails some risk, as infection and thrombosis might occur at the point of injection. In certain types of cases, however, where thrombosis is obviously present, I feel that this procedure would be justified and I propose to use it

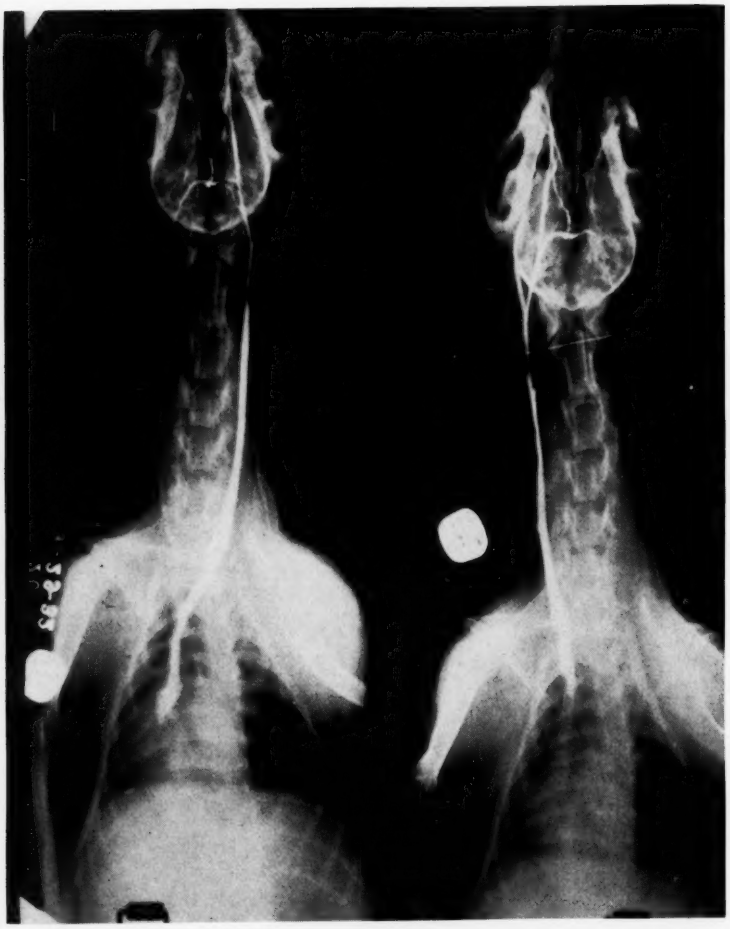


Fig. 5. X-ray of dog forty days following bilateral insertion of viable muscle implants in the external jugular veins. Five cubic centimeters of 50% sodium iodide was injected into the right and left sub-lingual veins. Metal cross bar marker shows the point of muscle insertion. Both veins have recanalized but still show a narrowing of the lumen at the point of muscle insertion. The left vein shows the characteristic retrograde constriction resulting from the thrombosis.

whenever the occasion might arise. I wish to here offer a word of warning in the use of sodium iodide intravenously. This substance produces a beautiful shadow, but in all the dogs in which it was used they all died as a result of the intravenous sodium iodide. It sets up a tremendous irritation of the vascular endothelium and they all developed an acute pulmonary inflammation with pleural effusion and died within thirty-six hours. I doubt very much if satisfactory studies can be made of the sigmoid sinus by the injection of neoskiodan into the carotid artery. Sodium iodide has been tried for the study of intracranial lesions with only fair success. The brain is so vascular that the overlying shadows are confusing. I have injected opaque media several times in the carotid artery of the dog and have been unable to get satisfactory venous shadows.

SUMMARY.

The viable muscle, on account of its rich supply of tissue juices, hastens the formation of a firm thrombus and plays an important part in the normal control of bleeding from an opened vessel. When viable muscle is applied to the opened sigmoid sinus it quickly unites to the vessel wall and aids in the healing process. As the muscle carries an additional blood and lymph supply to the wounded part it also aids in the control of the infection. Recanalization of the injured vein in the dog always occurs past the point of obstruction when viable muscle is used. The results of these experiments upon the dog lead me to believe that viable muscle also permits the recanalization and the normal restoration of function of the injured sigmoid sinus in the human.

I am deeply indebted to Dr. R. M. Isenberger, Professor of Pharmacology, University of Kansas, for valuable co-operation and in whose laboratory all of these experiments were conducted. I am also deeply obligated to Mr. Mark Carroll, my technical assistant, for many valuable suggestions. The x-ray studies were made in the laboratory of Dr. Galen Tice of the Department of Radiology, University of Kansas, and I wish to express my appreciation of his untiring assistance.

730 PROFESSIONAL BUILDING.

BIBLIOGRAPHY.

1. Mills, C. A.: Arch. Int. Med., 26:544, 1926.

2. Howell, Wm. H.: Coagulation of Blood. Textbook on Physiology, Vol. 12, 1933.

3. Morris (Quoted by Mills): Arch. Int. Med., 26:544, 1926.

4. Horsley, Sir Victor: Hemostasis by Application of Living Tissue. Brit. Med. J., 2:8, 1914.

LXXXVII.

THE INNERVATION OF THE NASAL MUCOSA, WITH SPECIAL REFERENCE TO ITS AFFERENT SUPPLY.*

KERMIT CHRISTENSEN, PH. D.,

ST. LOUIS.

Histologic investigations of the constituent neurones of the cranial autonomic ganglia, particularly the ciliary, have revealed multipolar ganglion cells of several morphologic types, and a limited number of bipolar and unipolar ganglion cells, the latter in some instances resembling sensory ganglion cells. According to current concepts of neurone structure and function, the multipolar neurones are indicative of the predominance of motor function in these ganglia. The bipolar or unipolar neurones, which resemble afferent ganglion cells in other parts of the nervous system, have been regarded by some as sensory in function.

Not infrequently sensory functions have been attributed to the autonomic system, particularly with reference to pain. Beginning with the work of Sluder, the sphenopalatine ganglion repeatedly has been regarded as the source of certain atypical pains in the head, on the assumption that lesions of this ganglion may give rise to pain. Relief of pain following anesthesia of the ganglion seemed to support the assumption that the sphenopalatine ganglion is not purely efferent. Statements in the recent literature concerning other parts of the autonomic nervous system also suggest the possibility of afferent conduction by autonomic neurones. For example, White (1933) has written: "The dictum that the autonomic is purely an efferent system concerned with motor control of the viscera and not directly concerned with visceral pain has been slowly discarded. The clinical and experimental observations of recent years have gradually forced neurophysiologists to give up this limited point of view and to admit that there are direct sympathetic pathways which carry pain sensations from the various viscera to the brain."

Results of certain physiologic experiments, in which the connections of autonomic ganglia with the central nervous system have

---

*From the Department of Microanatomy, St. Louis University School of Medicine.

been severed, have shown that stimulation of a peripheral area may elicit visceral reflexes. These results have been interpreted as supporting the assumption that peripheral sensory fibers may conduct impulses to sensory neurones located in an autonomic ganglion where synapses are made with sympathetic neurones (Schwartz, 1934).

In view of this tendency on the part of certain investigators to attribute sensory functions to autonomic nerves, it has seemed desirable to restudy microscopically suitable autonomic ganglia and the nerves connected with them in order to establish their anatomic relationships and thus provide an adequate anatomic basis for the interpretation of observed physiologic phenomena. The cranial autonomic ganglia in the cat with the nerves connected with them have afforded favorable material for this purpose. The present study has been devoted to the sphenopalatine ganglion and the nerves involved in the innervation of the nasal mucosa.

MATERIALS AND METHODS.

The sphenopalatine ganglion with its nerves and the nasal nerves from the orbit have been dissected, with the aid of a dissecting microscope whenever necessary, in adult cats, and from this material microscopic preparations have been made. Serial sections of the head of a full term fetus, 15 micra in thickness and stained with hematoxylin and eosin, have been used to determine some of the more minute anatomic relationships of the nerves.

Various silver methods have been used in efforts to obtain preparations of the ganglion cells with their processes. The methods which have yielded satisfactory results included the pyridine-silver method, the silver method of Cajal (McClung, 1929, Cajal method III) and a modified pyridine silver method (Gurdjian, 1927, method I), in which the tissues were treated with a chloral hydrate solution after the initial fixation.

Preparations of the nerves were made both by the pyridine silver method for staining axones of myelinated and unmyelinated fibers, and by the osmic acid method, for staining myelin sheaths of myelinated fibers. Paraffin sections were cut 6 to 8 micra in thickness.

In order to determine how many of the fibers in the nerves under investigation are sympathetic and how many are of other types, sections of these nerves also were studied from cats in which the superior cervical ganglion had been extirpated unilaterally, and sufficient time allowed for the degeneration of the sympathetic fibers extending upwards from it.

Lastly, certain of these nerves from cats which had been subjected to unilateral section of the roots of the upper four or five thoracic nerves just distal to the dorsal root ganglia were prepared by the Marchi method and studied for the number, extent and peripheral distribution of spinal nerve fibers extending into the head by way of the plexuses on the common and internal carotid arteries.

THE SPHENOPALATINE GANGLION.

Nerves to the Nasal Mucosa.—The sphenopalatine ganglion (Fig. 1) appears as an elongated, triangular, whitish body on the dorsal surface of the external pterygoid muscle just posterolateral to the sphenopalatine foramen, and between the median wall of the orbit and the infraorbital branches of the maxillary nerve. Of the nerves whose fibers enter the sphenopalatine ganglion, the nerve of the pterygoid canal is formed by the union of two nerves: the greater superficial petrosal, a branch of the seventh nerve, and the deep petrosal, a branch from the internal carotid plexus, the fibers of which arise chiefly in the superior cervical ganglion and ascend along the internal carotid artery. The nerve of the pterygoid canal passes forward on the surface of the external pterygoid muscle and joins the caudal and medial angle of the sphenopalatine ganglion. The sphenopalatine ganglion also is connected with the maxillary division of the trigeminal nerve through the sphenopalatine nerves. These nerves arise from the maxillary nerve, a short distance posterior to the ganglion, where several distinct bundles of fibers turn towards the ganglion, some of which unite with it. These bundles join the caudal and lateral angle of the ganglion, although in some cases bundles join the nerve of the pterygoid canal shortly before it reaches the ganglion. Fibers are conveyed from the sphenopalatine ganglion by the following groups of nerves, viz., the palatal, nasal, pharyngeal and orbital nerves. Only the nasal group will be considered in the present paper.

The posterior nasal and the nasopalatine nerves, which arise as two distinct trunks from the anterior apex of the ganglion, enter the nasal cavity through the sphenopalatine foramen. They are distributed to the mucosa, particularly of the ventrolateral and ventromedial parts of the nasal cavity, respectively. The lateral components pass forward along the ventrolateral aspect of the nose and terminate in the vascular connective tissue and in glandular tissue, particularly in a large compact mass of glandular tissue, which constitutes Stenson's gland. The medial component passes obliquely forward in the mucosa of the roof of the inferior meatus, then tends medianwards and continues forward along the ventral part of the nasal septum.

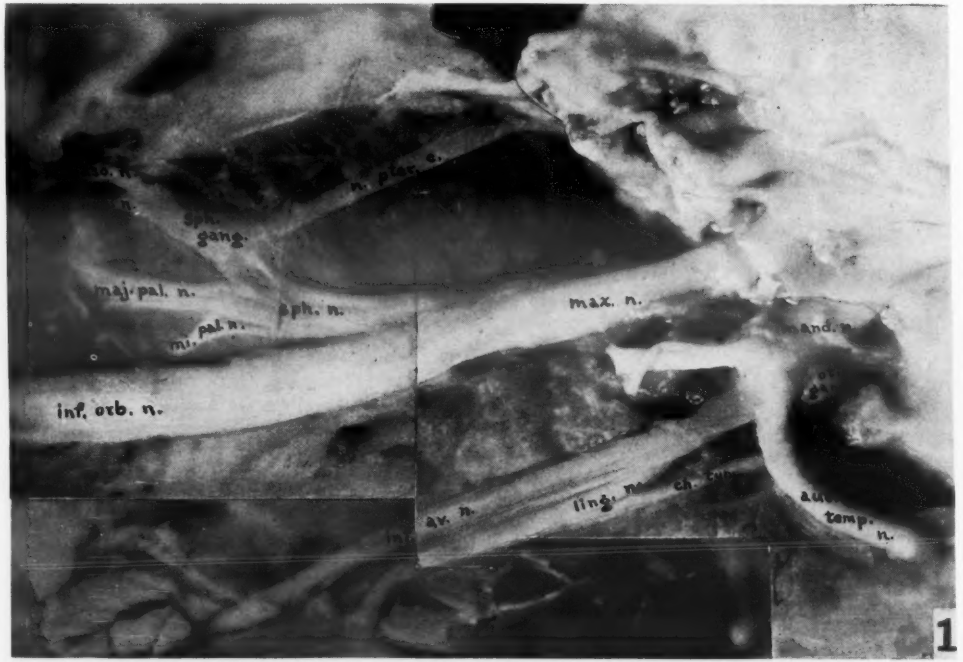


Fig. 1. Dissection of parts of the maxillary and mandibular nerves and of the sphenopalatine ganglion and its branches.

KEY

aur.-temp. n.—auriculotemporal nerve
ch. tymp.—chorda tympani
inf. av. n.—inferior alveolar nerve
inf. orb. n.—infraorbital nerve
l. nas. n.—lateral nasal nerve
ling. n.—lingual nerve
maj. pal. n.—major palatine nerve
mand. n.—mandibular nerve
max. n.—maxillary nerve
mi. pal. n.—minor palatine nerve
naso. n.—nasopalatine nerve
n. pter. c.—nerve of the pterygoid canal
sph. gang.—sphenopalatine ganglion
sph. n.—sphenopalatine nerves

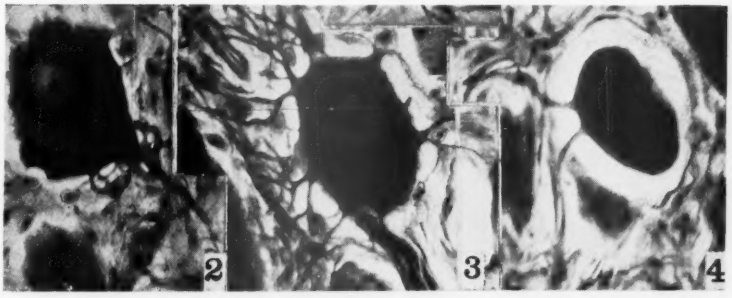
Some fibers pass upwards in the septal mucosa. The fibers of the nasopalatine nerves likewise are distributed to the vascular and glandular tissue.

The nasociliary nerve, arising in the orbital fissure as a branch of the ophthalmic nerve, can be traced obliquely forward across the orbit to a position above the optic nerve, where the nasociliary nerve divides into two branches, the ethmoid nerve and the infratrochlear nerve. The ethmoid nerve passes through the ethmoid foramen and eventually its fibers are distributed to both lateral and medial surfaces of the nasal mucous membrane superior to the parts supplied by the nasal nerves from the sphenopalatine ganglion.

Morphology of Sphenopalatine Ganglion Cells.—According to the accounts of various investigators, including Retzius, 1880; von Lenkossek, 1894; Muller and Dahl, 1910; Carpenter, 1912; Larsell and Fenton, 1928, and Slavich, 1933, the great majority of the ganglion cells in the sphenopalatine ganglion are multipolar neurones. Most of these have dendrites that extend for some distance beyond the capsule (Fig. 3), although some have both extracapsular and minute endocapsular dendrites. As observed in our preparations, the extracapsular dendrites usually are relatively small and may or may not branch profusely. Occasionally, cells with long, moderately heavy dendrites, having few branches, are found. Fenestrated cells (Fig. 2) also occur in the sphenopalatine ganglion. The fenestrations appear as short, slender, profusely branched, anastomosing processes, often located on one side of an elongated, angular cell. Not uncommonly the axone arises from this fenestrated area. In some cells, the fenestrations are less complex and consist merely of several loops.

In rare instances, a cell has distinct double processes. The cell illustrated in figure 4 resembles a developing sensory ganglion cell in the bipolar stage. Some cells exhibit a single large process. These resemble unipolar neurones, but they and the neurones resembling bipolar cells usually exhibit a number of delicate processes arising from the cell body; consequently, they probably must be regarded as multipolar. In general they conform to the descriptions, particularly of Pines and his associates, of certain cells in the ciliary ganglion which have been regarded as afferent in function.

As stated above, the great majority of the sphenopalatine ganglion cells are typically multipolar. The so-called unipolar and bipolar neurones are present in such small numbers that even if they should have to be regarded as sensory, this ganglion could not be regarded as an important sensory center. These cells, furthermore, exhibit minute processes in addition to the larger processes. In this respect



Morphologic types of sphenopalatine ganglion cells. Pyridine silver method with chloral hydrate.

Fig. 2. Fenestrated cell.

Fig. 3. Multipolar cell with moderately long, branched dendrites.

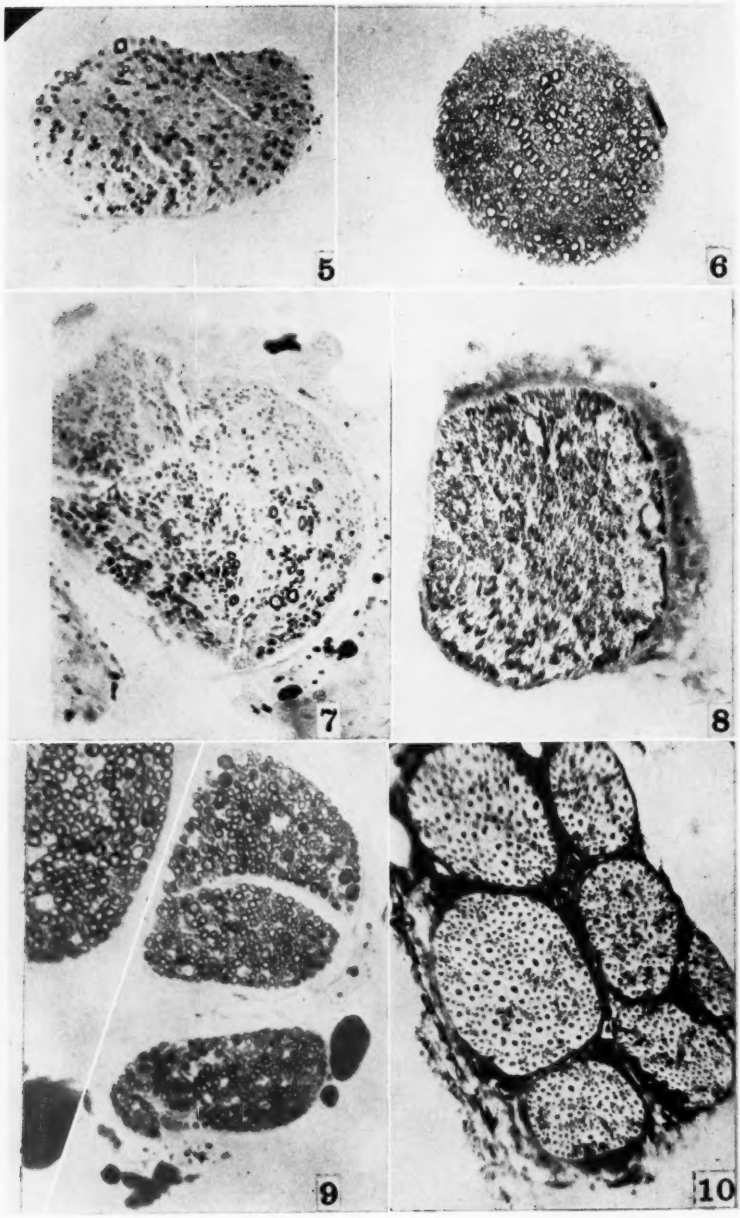
Fig. 4. A cell with bipolar characteristics.

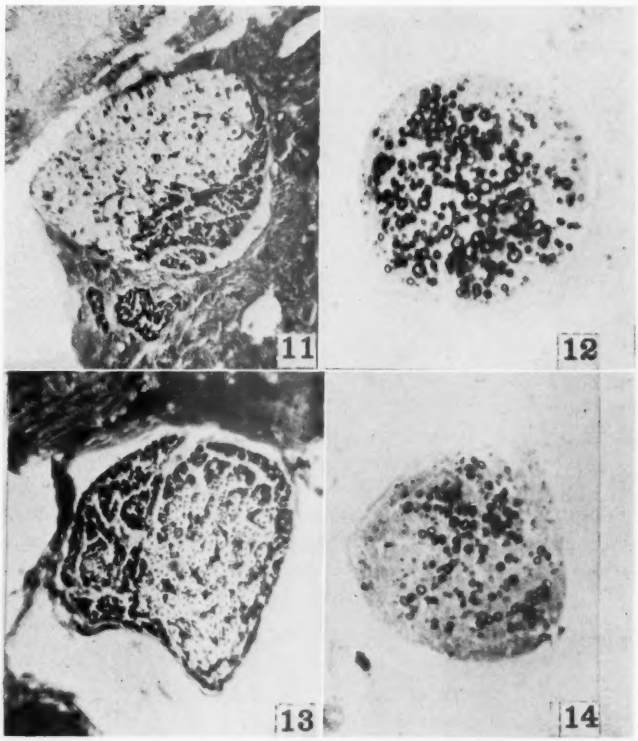
they differ from the peripheral neurones, which are known to be sensory. The evidence, therefore, does not support the assumption that this ganglion includes afferent neurones, and it must be concluded that the nerves connected with it convey sensory fibers only that arise in other sources.

The Nerves Connected with the Sphenopalatine Ganglion.—The nerve of the pterygoid canal is the pathway through which the sphenopalatine ganglion receives preganglionic and sympathetic fibers. Since it is formed by a union of the greater superficial petrosal nerve (carrier of the preganglionic fibers) and the deep petrosal nerve (carrier of sympathetic fibers from the internal carotid plexus), these two nerves will be considered first.

The greater superficial petrosal nerve (Fig. 6) includes a number of moderately large, thickly myelinated fibers (diameter, 10 micra), some of medium sizes (diameter, 6 micra) and many small myelinated fibers (diameter, about 2 micra). The number of the large myelinated fibers varies in different animals, some having only a few, others having a considerable number. The nerve also contains a few unmyelinated fibers. The small myelinated fibers are considered to be preganglionic. The deep petrosal nerve (Fig. 5) has small myelinated fibers scattered in the section and only a few large myelinated fibers. The bulk of the section is occupied by unmyelinated fibers, which are sympathetic (see also Larsell and Fenton, 1928, and Burns and Larsell, 1931).

The nerve of the pterygoid canal represents a composite of the fibers from the two nerves named above; consequently it includes


5
6
7
8
9
10



NERVES OF THE SPHENOPALATINE GANGLION

Fig. 5. Deep petrosal nerve. Osmic acid method. Shows number, size and distribution of myelinated fibers. The position of the unmyelinated fibers is suggested in the unstained areas. Diameter of large fibers, 10 micra; small fibers, 2.5 to 2 micra.

Fig. 6. Greater superficial petrosal nerve. Osmic acid method. Myelinated fibers demonstrated. Diameter of large fibers, 10 to 8.5 micra; medium sized fibers, 6 micra; smallest fibers, 1.5 micra.

Fig. 7. Nerve of the pterygoid canal. Osmic acid method. Size, number and distribution of myelinated fibers shown. The number of the large myelinated fibers in various nerves of the pterygoid canal exhibits an individual variation. Diameter of large fibers, 10 micra; medium sized fibers, 6 micra; smallest fibers, 2.5 to 1.5 micra.

Fig. 8. Nerve of the pterygoid canal. Pyridine silver method. An abundance of unmyelinated fibers are present.

Fig. 9. Sphenopalatine nerves. Osmic acid method. Myelinated fibers predominate. Diameter of large fibers, 10 to 12 micra; medium sized fibers, 6 micra; small fibers, 2.5 to 1.5 micra.

Fig. 10. Sphenopalatine nerves. Pyridine silver method. Some unmyelinated fibers are shown to be present.

Fig. 11. Lateral nasal nerve. Pyridine silver method. Demonstrates the unmyelinated fibers, some of which are sympathetic from the superior cervical ganglion, others parasympathetic from the sphenopalatine ganglion.

Fig. 12. Lateral nasal nerve. Osmic acid method. The types of myelinated fibers that have been distributed by way of the sphenopalatine ganglion are shown. Diameter of large fibers, 10 micra; medium sized fibers, 5 micra; small sized fibers, 2.5 to 2 micra.

Fig. 13. Nasopalatine nerve. Pyridine silver method. Shows again the number of unmyelinated fibers present, which are similar in type to those in the lateral nasal nerve.

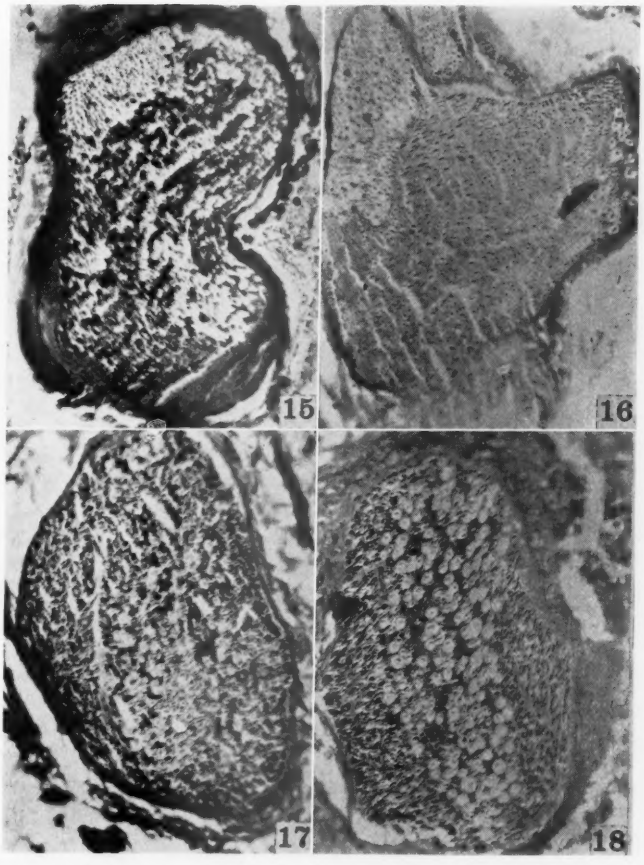
Fig. 14. Nasopalatine nerve. Osmic acid method. Again, types of myelinated fibers distributed by way of the sphenopalatine ganglion are shown. Diameter of large fibers, 10 to 8 micra; small fibers, 2.5 to 2 micra.

moderately large (diameter, 10 micra), medium sized (diameter, 6 micra) and small (diameter, 2 microns) myelinated fibers (Fig. 7), the small ones being the most numerous. The few large myelinated fibers in the nerve of the pterygoid canal illustrated suggest that only a few fibers of this size must have been present in the greater superficial petrosal nerve. The preganglionic fibers are represented by the many small myelinated fibers seen in sections of the greater superficial petrosal nerve and the nerve of the pterygoid canal (Larsell and Fenton, 1928). Sections of the nerve of the pterygoid canal in some cases show many more myelinated fibers, at least close to the ganglion, than are shown in the section illustrated. The additional myelinated fibers, in such cases, come from rami of the sphenopalatine nerve which join the nerve of the pterygoid canal just before it reaches the ganglion. The section of the nerve of the pterygoid canal illustrated in figure 8 (pyridine silver method) demonstrates the abundance of unmyelinated fibers which come mainly from the deep petrosal nerve.

The sphenopalatine nerves which join the sphenopalatine ganglion and are described as its sensory roots, convey fibers which traverse the ganglion and enter the nerves which arise from it. The sphenopalatine nerves are made up chiefly of large (diameter, 10 to 12 micra), medium sized (diameter, 6 micra) and small (diameter, around 2 micra) myelinated fibers (Fig. 9). Pyridine silver preparations, however, show areas in which unmyelinated or thinly myelinated fibers are present (Fig. 10). It has been suggested that some sympathetic fibers from the internal carotid plexus join the maxillary nerve and thus reach the sphenopalatine ganglion through the sphenopalatine nerves. If this be true, some of the unmyelinated fibers in the sphenopalatine nerves are sympathetic.

The nerves described above which convey fibers to the sphenopalatine ganglion are made up in part of fibers which effect synaptic connections there and in part of fibers which merely traverse the ganglion and enter the nerves that arise from it. The nerves which are given off by the sphenopalatine ganglion, therefore, are composites of fibers which originate in the ganglion and fibers which pass through it. The nerve fibers arising in the sphenopalatine ganglion are mainly unmyelinated and account for a portion of the unmyelinated fibers in the nerves distributed to the nasal mucous membrane (Langley, 1898).

Sections of the posterior lateral nasal nerve (Fig. 12) and of the nasopalatine nerve (Fig. 14) show that each of these nerves contains some moderately large and some small myelinated fibers. Pyridine



Sections of the nerves connected with the sphenopalatine ganglion, following extirpation of the superior cervical ganglion. Changes in the number of unmyelinated fibers on the operated side are to be observed.

Fig. 15. Nerve of the pterygoid canal from the unoperated side. Pyridine silver method.

Fig. 16. Nerve of the pterygoid canal from the operated side. Pyridine silver method.

Fig. 17. Lateral nasal nerve from the unoperated side. Pyridine silver method.

Fig. 18. Lateral nasal nerve from the operated side. Pyridine silver method.

silver sections of these nerves (Fig. 13) also exhibit numerous unmyelinated fibers. The myelinated fibers come from the nerve of the pterygoid canal and the sphenopalatine nerves. The unmyelinated fibers which do not have their origin in the sphenopalatine ganglion (parasympathetic) are largely sympathetic fibers from the superior cervical ganglion which come by way of the nerve of the pterygoid canal.

The effects of removal of the superior cervical ganglion upon the nerves described above are particularly striking. Osmic acid preparations of the nerve of the pterygoid canal on the operated side do not differ apparently from similar sections taken from the control side. Pyridine silver preparations of the nerve of the pterygoid canal on the operated side (Fig. 16), however, show very definitely that a large number of the unmyelinated fibers have undergone degeneration. There remain within the nerve only a few unmyelinated fibers, besides the small and larger myelinated fibers. These myelinated fibers represent the sensory and the preganglionic fibers.

A section of the posterior lateral nasal nerve, from the operated side (Fig. 18), likewise exhibits degeneration of a part of the unmyelinated fibers. The unmyelinated fibers which remain intact are mainly the postganglionic parasympathetic fibers which come from the sphenopalatine ganglion. No perceptible differences in the myelinated fibers of operated and unoperated sides can be detected in sections prepared by the pyridine silver method.

The evidence at hand seems to support the opinion that the myelinated fibers which traverse the ganglion and are distributed through the nasal nerves are mainly sensory. The larger myelinated fibers in these nerves undoubtedly are sensory. What proportion of the smaller myelinated and unmyelinated components of these nerves which continue distalwards in the nasal nerves are sensory cannot be determined at this time. The few large myelinated fibers in the nerve of the pterygoid canal, regarded as sensory, were early described by Langley (1898), and their presence in the greater superficial petrosal nerve has been noted by Larsell and Fenton (1928). Yagita (1914) advanced evidence that sensory fibers exist in the nerve of the pterygoid canal, based on the demonstration of chromatolysis in cells of the geniculate ganglion following section of this nerve. That either myelinated or unmyelinated sensory fibers reach the nerve of the pterygoid canal in considerable numbers by way of the deep petrosal nerve seems improbable.

Physiologic investigations over a period of years have served to establish the function of the sphenopalatine ganglion and its nerves.

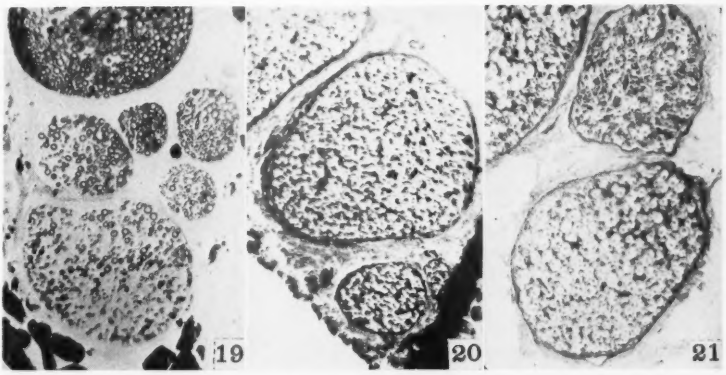


Fig. 19. Nasociliary nerve. Osmic acid method. The myelinated fibers are scattered among unmyelinated fibers, except in the branch which is the infratrochlear nerve. Many of the myelinated fibers in the ethmoid nerve are small. Diameter of large fibers, 13 micra; small fibers, 2 to 4 micra.

Fig. 20. Nasociliary nerve. Pyridine silver method. Shows the position of the unmyelinated fibers. This section is taken from the unoperated side of the same animal as the section in figure 21.

Fig. 21. Nasociliary nerve. Pyridine silver method. The nerve is from the side from which the superior cervical ganglion was removed. Not all of the unmyelinated fibers degenerate.

By electrical stimulation of the sphenopalatine ganglion cells, Prevost (1866) demonstrated vasodilator fibers to the nasal mucosa. Langley (1898) found that vasodilator and also secretory fibers from the sphenopalatine ganglion reach not only to the mucosa of the nose but also to the soft palate, the tonsils, the uvula, the roof of the mouth, the upper lip and upper gums, and the upper part of the pharynx. Tschallussow (1913) described the vasomotor pathway to the nasal mucous membrane as consisting of preganglionic fibers from the facial nerve which pass to the sphenopalatine ganglion and synapse there with neurones from which the postganglionic fibers arise.

On the basis of a physiologic study of the sphenopalatine ganglion and the nerves to the nose in the dog, Blier (1930) concluded that vasomotor pathways reaching the nose through the nerve of the pterygoid canal involve (1) preganglionic fibers coming from the seventh nerve by way of the greater superficial petrosal nerve which effect synaptic connections with neurones in the sphenopalatine ganglion and (2) a few sympathetic vasoconstrictor fibers coming from the superior cervical ganglion through the deep petrosal nerve. Other vasomotor fibers, particularly vasoconstrictors, which he regarded as

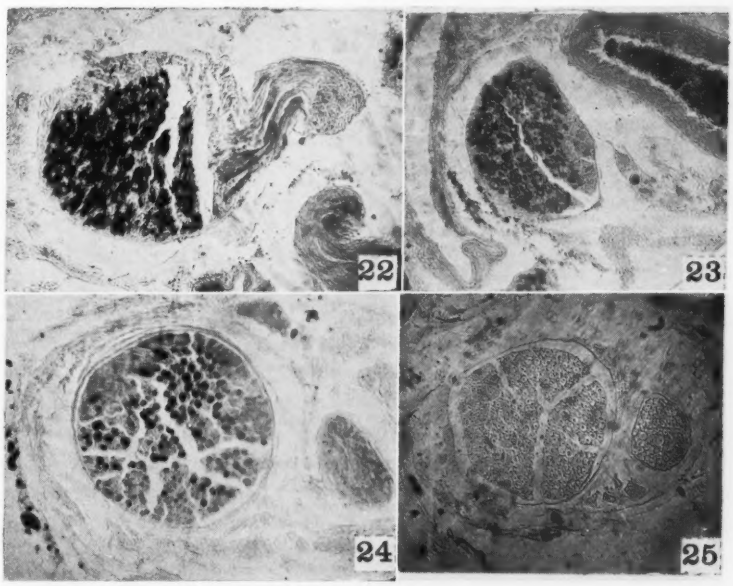
sympathetic and as arising in the superior cervical ganglion, traverse the internal carotid nerve to the maxillary nerve, through which they reach the sphenopalatine ganglion. The sensory fibers are mainly afferent components of the trigeminal and facial nerves. Stimulation of the central end of the nerve of the pterygoid canal, according to Blier, resulted in lingual, facial, ocular, respiratory and cardiac responses. He, however, did not regard his findings as affording an explanation of the reported results of cocainizing the sphenopalatine ganglion in man.

The Nerves from the Orbit.—Sections through the nasociliary nerve (Fig. 19), of which the ethmoid nerve is a branch, show it to be an intermixture of myelinated and unmyelinated fibers, the unmyelinated fibers (Fig. 20) of which form a conspicuous part of each ramus except the infratrochlear nerve, in which myelinated fibers predominate. The infratrochlear nerve contains more of the large myelinated fibers (diameter, 12 micra) than other branches. The small myelinated fibers (diameter, 2 to 4 micra) and fibers of medium size (diameter, 6 to 8 micra) are more in evidence in the branches which include the ethmoid nerve. The ethmoid nerve carries to the nose mainly the myelinated fibers of lesser diameter and unmeylinated fibers.

Extirpation of the superior cervical ganglion results in no apparent change in the number of myelinated fibers in the nasociliary nerve, but in a material reduction in the number of unmyelinated fibers (Fig. 21).

The fibers in the nasociliary nerve are either sensory or sympathetic. A part of the unmyelinated fibers have their origin in the superior cervical sympathetic ganglion, pass into the plexus on the internal carotid artery, and from there reach the ophthalmic nerve. These fibers disappear following removal of the superior cervical ganglion. The large myelinated fibers are undoubtedly sensory, and Windle (1926) concluded that the small myelinated and unmyelinated fibers remaining after removal of the superior cervical ganglion also are sensory. They arise mainly in the semilunar ganglion.

In addition to the afferent fibers reaching the nasal mucosa from the courses pointed out above, Kuntz (1934) has shown that fibers of spinal and vagus origin are associated with the cephalic autonomic nerves. In Marchi preparations, following section of the roots of the upper thoracic nerves, he observed degenerated myelinated fibers in very considerable numbers in the nerves associated with the common carotid and the proximal portions of the internal and external carotid



Sections of the nasociliary and nasal nerves after unilateral section of upper four or five thoracic nerves just distal to spinal ganglion. Marchi method.

Fig. 22. Nasal nerve. Operated side.
Fig. 23. Nasal nerve. Unoperated side.
Fig. 24. Nasociliary nerve. Operated side.
Fig. 25. Nasociliary nerve. Unoperated side.

arteries. Since preganglionic nerve components which extend cephalad from the upper thoracic segments, according to the best evidence, terminate in the superior cervical ganglion, the myelinated fibers which undergo degeneration in the plexuses on the carotid arteries following section of the roots of the upper thoracic nerves must be regarded as afferent components of these nerves. In order to determine whether these fibers extend distalwards as far as the nasal mucosa, Marchi preparations of the nasal nerves from the sphenopalatine ganglion and of the nasociliary nerve from animals in which the upper four or five thoracic spinal nerves had been sectioned unilaterally just distal to the dorsal root ganglia have been studied. Sections of the nasal nerve (Fig. 22) exhibit degeneration of a considerable number and sections of the nasociliary nerve (Fig. 24) of a lesser number of myelinated fibers. A third source of afferent fibers reaching the nasal mucosa, some of which must pass through the sphenopalatine ganglion, therefore, is demonstrated.

DISCUSSION.

One of the chief aims of this study has been to discover whether there is any morphologic evidence which would support the assumption that the autonomic nervous system, particularly certain cranial autonomic ganglion cells, play a rôle in afferent or sensory conduction. The results warrant only a negative conclusion on this point. The ganglion cells which resemble sensory ganglion cells most closely occur in the ciliary and sphenopalatine ganglia. These ganglion cells which, disregarding their minute cytoplasmic processes, resemble unipolar and bipolar cells, respectively, are so few that even though they should be unmistakably sensory, they could hardly form important sensory centers. In the development of autonomic ganglion cells, according to DeCastro (1932), differentiating neuroblasts first have a single process, later two, and finally many. It may be assumed, therefore, that the ganglion cells with one or two large processes, observed in certain of the cranial autonomic ganglia, have become arrested at the unipolar or bipolar stage of differentiation. There is no evidence that these cells differ in function from the multipolar ganglion cells. As has been pointed out above, furthermore, the so-called unipolar and bipolar ganglion cells observed in the ciliary and sphenopalatine ganglion exhibit numerous minute processes; consequently, they probably should be regarded as multipolar.

A study of the nerves connected with the sphenopalatine ganglion affords no evidence in support of the assumption that any sensory fibers arise in this ganglion. The results of this investigation seem to warrant the conclusion that the sensory functions, which have been attributed to this ganglion are in reality the functions of sensory fibers which traverse it and the nerves with which it is connected. Of all the cranial autonomic ganglia, the sphenopalatine is the most important in this regard, because it is associated anatomically with sensory fibers arising not only in the semilunar ganglion of the fifth but also in the geniculate ganglion of the seventh cranial nerve. These fibers pass through the sphenopalatine ganglion and become components of the nerves connected with it. Some of the afferent components of the vagus and upper thoracic nerves which ascend in the plexuses on the common and internal carotid arteries (Kuntz, 1934), also traverse the sphenopalatine ganglion and reach the nasal mucosa by way of the nerves connected with it. The sphenopalatine ganglion, therefore, is traversed by sensory fibers of various origins. The sensory functions which have been attributed to it obviously are functions of the sensory fibers. The ganglion cells in the cranial autonomic ganglia, probably without exception, constitute the final

links in efferent conduction pathways; consequently, they are essentially efferent.

Whatever success may have been achieved in the treatment of sensory disturbances by anesthetizing the sphenopalatine ganglion probably can be best explained on the assumption that the sensory fibers passing through the ganglion have been blocked and that intervention at this point has been advantageous because the ganglion represents the point of concentration of the sensory fibers which traverse the nerves connected with it. The data set forth in the present paper do not support the clinical suggestion that irritation of the ganglion is the direct cause of afferent impulses.

SUMMARY AND CONCLUSIONS.

1. The sphenopalatine ganglion has been studied for morphologic types of ganglion cells (silver methods). The nerves connected with this ganglion and the nasociliary nerve have been analyzed with reference to their constituent unmyelinated (pyridine silver method) and myelinated fibers (osmic acid method).

2. The sphenopalatine ganglion cells are mainly multipolar. Occasional ones have one or two conspicuous processes, but show very minute cytoplasmic processes; consequently, they probably should be regarded as multipolar and efferent in function.

3. The nerve of the pterygoid canal, which conveys preganglionic fibers to the sphenopalatine ganglion, represents a composite made up of some large and medium sized and many small myelinated fibers from the greater superficial petrosal nerve and a few myelinated and many unmyelinated fibers from the deep petrosal nerve. The unmyelinated fibers are mainly sympathetic, since they degenerate following removal of the superior cervical sympathetic ganglion. The small myelinated fibers are mainly preganglionic. The remaining fibers are afferent, having their origin mainly in the geniculate ganglion.

4. The sphenopalatine nerves convey many myelinated and a few unmyelinated fibers to the sphenopalatine ganglion. The myelinated fibers are sensory and have their origin in the semilunar ganglion. The unmyelinated fibers probably are mainly sympathetic.

5. The lateral nasal and nasopalatine nerves carrying fibers from the sphenopalatine ganglion are made up of large, medium sized, and small myelinated and many unmyelinated fibers. The unmyelinated fibers are in part parasympathetic fibers arising in the sphenopalatine ganglion and in part sympathetic fibers which traverse it. The myelinated fibers are mainly afferent components of the fifth and seventh nerves which traverse the sphenopalatine ganglion.

6. The nasociliary nerve through its ethmoid branch supplies large, medium sized and small myelinated and unmyelinated fibers to the nasal mucosa. Some of the unmyelinated fibers are sympathetic since they undergo degeneration following extirpation of the superior cervical sympathetic ganglion. The remaining fibers, including unmyelinated, small myelinated, and, in a few instances, larger myelinated fibers, probably are sensory and have their origin mainly in the similunar ganglion.

7. The nasal nerves from the sphenopalatine ganglion and the nasociliary nerve also include some afferent components of the vagus and the upper thoracic spinal nerves. For the latter nerves this is indicated by the presence of degenerated myelinated fibers in Marchi preparations of the nasal and nasociliary nerves following section of the upper thoracic nerves.

8. The anatomic findings set forth in this paper do not support the assumption that autonomic ganglia include afferent or sensory ganglion cells, but in general support the theory that the ganglion cells in the cranial autonomic ganglia, like those in the ganglia of the sympathetic trunks, are essentially efferent. The afferent functions which have been attributed to certain of the cranial autonomic ganglia, particularly the sphenopalatine, can be explained satisfactorily on the basis of the afferent fibers which traverse them.

The writer wishes to express his appreciation to Dr. Albert Kuntz for helpful advice and criticism throughout the preparation of this paper.

BIBLIOGRAPHY.

Blier, Z.: Physiology of the Sphenopalatine Ganglion. Am. J. Physiol., 93:398, 1930.

Burger, H.: Das Ganglion Sphenopalatinum. Acta Otolaryngol., 11, 222, 1927.

Burns, E. M., and Larsell, O.: Myelinated Fibers in the Vidian Nerve and Its Connections. Abst. Anat. Rec., 48:39, 1931.

Carpenter, F. W.: On the Histology of the Cranial Autonomic Ganglia of the Sheep. J. Comp. Neur., 22:447, 1912.

Chorobski, J., and Penfield, W.: Cerebral Vasodilator Nerves and Their Pathway from the Medulla Oblongata with Observations of the Pial and Intracerebral Vascular Plexus. Arch. Neur. and Psychiat., 28:1257, 1932.

deCastro, F.: Sympathetic Ganglia, Normal and Pathologic: Cytology and Cellular Pathology of the Nervous System. Edited by Wilder Penfield. Paul B. Hoeber, New York. 1932.

Gurdjian, E. S.: The Diencephalon of the Albino Rat. J. Comp. Neur., 43:1, 1917.

Jung, L., Tagand, R., and Chavanne, F.: Etude experimentale du rôle secrétoire et de rôle trophique des elements nerveux du carrefour spheno-palatin. Oto-rhino-laryngol. Internatal. 10:353, 1926.

Klepper, J. I.: Demonstration of Specimens of the Sphenopalatine Ganglion. Laryngoscope, 38:41, 1928.

Kuntz, A.: The Autonomic Nervous System. Lea & Febiger, Philadelphia, 1934.

Kuntz, A.: Nerve Fibers of Spinal and Vagus Origin Associated with the Cephalic Sympathetic Nerves. ANNALS OF OTOLOGY, RHINOLOGY AND LARYNGOLOGY, 43:50, 1934.

Kuré, K., and Sakurasawa, F.: Ueber die parasympathische Fasern für das Ganglion sphenopalatinum und über den Verlauf Sekretionsfasern für die Tranendrüse. Zeit. für Zellforsch. und Mikr. Anat., 9:245, 1929.

Larsell, O., and Fenton, R. A.: The Embryology and Neurohistology of Sphenopalatine Ganglion Connections: A Contribution to the Study of Otalgia. Laryngoscope, 36:371, 1928.

v. Lenhossek, M.: Ueber das Ganglion sphenopalatinum und die sympathischen Ganglien. Beitrage zur Histologie des Nervensystems und der Sinnesorgane. Wiesbaden, s. 163, 1894.

McClung, C. E. (Editor): Handbook of Microscopical Technic. Neurological Technic by W. H. F. Addison, p. 317. Paul B. Hoeber, New York, 1929.

Müller, L. R.: Die Lebensnerven. Julius Springer, Berlin, 1924.

Müller, L. R., and Dahl, W.: Die Beteiligung des sympathischen Nervensystems an der Kopfinnervation. Deutsch. Arch. f. klin. Med., 99:48, 1910.

Pines, J. L.: Zur Morphologie des Ganglion ciliare beim Menschen. Zeit. f. mikr-anat. Forsch., 10:313, 1927.

Pines, J. L., and Friedman, E.: Zur vergleichenden Histologie des Ganglion ciliare bei Säugetieren. Zeit. f. mikr. anat. Forsch., 16:259, 1929.

Rhinehart, D. A.: The Nervus Facialis of the Albino Mouse. J. Comp. Neur., 30:81, 1918.

Rossi, O.: On the Afferent Paths of the Sympathetic Nervous System, with Special Reference to the Nerve Cells of Spinal Ganglia Sending Their Peripheral Processes into the Rami Communicantes. J. Comp. Neur., 34:493, 1922.

Schwartz, H. G.: Observations on the Reflex Activity in the Sympathetic Nervous System. Abst. Anat. Rec., 58:36, 1934.

Stewart, D., and Lambert, V.: The Sphenopalatine Ganglion. J. Laryngol. and Otol., 45:753, 1930.

Slavich, E.: Confronti fra la morfologia di gangli del parasympatico encefalico e del simpatico cervicale con speciale riguardo alla struttura del ganglio ciliare. Zeit. fur Zellforsch. und Mikr. Anat., 15:688, 1932.

Sluder, G.: Nasal Neurology, Headaches and Eye Disorders. C. V. Mosby Co., St. Louis. 1927.

Tanaka, T.: Ganglion sphenopalatinum des Menschen. Heft 3, Nr. 87, s. 91. Arbeit. Anat. Inst. Kaiser, Univ. Kyoto Serie A. 1932.

Terracol, J.: Le ganglion sphenopalatin. Archiv. de Laryngol., Otol. et de Rhin., 4:787, 1925.

Tschallussow, M. A.: Die Innervation der Gefässe der Nasenschleimhaut. Pflüger's Arch., 151:523, 1913.

Vogel, K.: Die Beziehungen des Ganglion sphenopalatinum zu den von ihm ausgehen Nervenstämchen. Zeit. für Hals- Nasen- und Ohrenheilkunde, 25:485, 1931.

White, J. C.: Progress in the Surgery of the Sympathetic Nervous System in 1932. New Engl. J. Med., 209:843, 1933.

Windle, W. F.: The Distribution and Probable Significance of Unmyelinated Nerve Fibers in the Trigeminal Nerve of the Cat. J. Comp. Neur., 41:453, 1926.

Yagita, K.: Einige Experimente in dem Nervus petrosus superficialis major zur Bestimmung des Ursprungsgebietes des Nerven. Folia Neurobiologica, 8:361, 1914.

LXXXVIII.

CAVERNOUS SINUS THROMBOSIS: WITH RECOVERY, PROVED BY NECROPSY.

EUGENE R. LEWIS, M. D.,

LOS ANGELES.

A case presenting clinical picture of thrombosis of cavernous sinus, staphylococcus aureus bacteremia, toxic encephalitis, extensive acute cranial and spinal meningitis affecting II, III, IV, V, VI, VII, VIII cranial nerves recovered general physical, nervous and mental levels, except for altered reflexes, Babinski, impaired vestibular, pupillary, left abducens and left ocular nerve functions; on discharge this pathologic episode had apparently closed. Three years later he died suddenly of acute right tympanomastoiditis with intracranial complications; necropsy not only revealed cause of death but confirmed previous diagnosis of infectious left cavernous sinusitis with recovery.

REPORT OF CASE.

CASE 1.—R. S., schoolboy, 15 years old: nothing significant elicited in family history; considered strong and well; the last week of October, 1930, developed a "boil" or "large pimple" on left cheek, near angle of mouth; following incision the cheek became swollen and painful; after still further incisions, swelling and pain increased, involving face, eyelids, scalp and retroauricular cervical regions. On admission to Hollywood Hospital next day found to have staphylococcus aureus in blood, leucocytes and albumin in urine; no leucocyte or erythrocyte count was made, bacteriophage was given intravenously and patient transferred to Los Angeles General Hospital.

Notes on entrance: "Unconscious; temperature 103°-104°; 34,000 leucocytes, 94 per cent polys; spinal fluid 60 cells, neck stiff, both eyeballs proptosed and intense chemosis of conjunctivæ and lids. Diagnosis: Cavernous sinus thrombosis; examination incomplete; an early fatal termination expected."

Excerpts from clinical chart: "Nov. 21st, apparently moribund, partially conscious, both eyeballs proptosed—extreme bilateral ophthalmoplegia. Nov. 24th: Conscious, neck markedly stiff, not in pain but generally hyperesthetic; especially sensitive in upper cheeks and forehead, arms, body and legs; complete paralysis both VI, eye movements greatly impaired; chemosis and proptosis of eyeballs, right eye central vision good, media clear, red disc, outlines sharp, cup details obscured, no vascular lesions; left eye blind, media clear, disc margins unclear, cup details obscured, peripapillary grayish exudate, no vascular lesions; I, anosmia?; manifest impairments of II R and L., III, IV, VI R. and L., V upper div. R, VII R. (facial lag), VIII (cochlear and vestibular); personality changes—pettish, apprehensive, unco-operative; request neurologic examination and complete spinal fluid examination."

Neurologic findings (Drs. Ingham and Neilson), Nov. 25th: "Partially conscious, both eyeballs proptosed, almost complete bilateral ophthalmoplegia; anosmia; no pains but generally hyperesthetic, especially in upper cheeks and forehead, more on the right; hyperesthetic areas on arms and legs; degree of paralysis of each abducens nerve difficult to evaluate because of ophthalmoplegia, proptosis and chemosis; right visual acuity good, left dull; media clear; disc red, outlines sharp, cup details obscured; no vascular changes; left disc details obscured, peripapillary exudate, no vascular changes." Dec. 3rd: "Meningitis with multiple cranial nerve damage secondary to cellulitis of the face; still unconscious; spinal fluid content 750 cells per cu. cm., 80 per cent polymorphs." Dec. 8th: "Universal tenderness, cloudy mental state, co-operation insufficient for complete examination; would not permit eyes to be touched; left eye blind, pupil reacts consensually but not to light; optic atrophy apparently complete but vestige of light perception found in left temporal periphery; no apparent impairment of right visual acuity; pupil reacts to light but not consensually or on convergence; bilateral impairment III and VI nerves; bilateral ptosis, more marked right; Babinski left, bilateral Kernig, abdominal reflexes normal; marked rigidity of neck." Summary: "Diffuse infection beginning as cellulitis of the face, intracranial extension with basilar meningitis and cavernous sinus thrombosis; apparently recovering." January 16th, 1931: "Spinal puncture 1,700 cells—80 per cent polymorphs; 15 cc. 1 per cent mercurochrome intravenously." Three spinal punctures during February showed decreasing cell counts. March 16th, "residuals present, but no evidence of activity; total blindness, left, with complete optic atrophy; both pupils react on convergence; slight right ptosis; left VI partially impaired; deep reflexes active, upper extremities spastic; positive Babinski, Chaddock and Gordon reflexes on left."

During November temperature ranged from 101°-104°; frank meningitis, opisthotonos, exophthalmos, chemosis, exquisite hypersensitiveness, dermagraphia, marked personality changes and bulimia; euphoric and garrulous during late November and early December, with higher temperatures (central hyperthermia); spinal fluid in December contained 300 cells, 200 cells, 200 cells.

Between Christmas and New Year's return of function noted in right VI, later in left VI; leukocytosis continued—10,000 and 20,000—until January when gradual subsidence began, coincident with returning normal personality, appetite and sleep; gained 12 lbs. between December 15th and February 21st; February 1st began walking with considerable difficulty; frequent examinations revealed central cochlear and vestibular impairments attributable to left supretentorial lesions; between February 21st and April 7th gained 3 lbs.; left eye remained blind and both discs showed secondary atrophic changes of retrobulbar nature.

Review.—(1) Acute infection in drainage area of left facial and angular veins; (2) before being seen repeated incisions had been made, with ensuing local exacerbations general febrile symptoms; (3) intracranial extensions, cerebral and spinal meningitic symptoms, gross vascular changes with bilateral proptosis, intense lid and retrobulbar chemosis; marked disturbance of forebrain functions; (4) febrile reactions with leucocytosis; turbid spinal fluid with high cell count; hyperthermia, basal meningitis, bulimia, dermagraphia, body-weight and personality changes; (5) gradual general and local improvements, with restoration of personality, body weight and (incomplete) II, III, IV, V, VI, VII, VIII nerve functions.

After going home, April 7th, he resumed school work, completing the term without interruption. In August, 1931, several "boils" developed, discharging for a number of weeks with no ill effects. Throughout next year he walked four miles to and from school daily, felt, ate and slept well, missed no school, and did

Fig. 1.

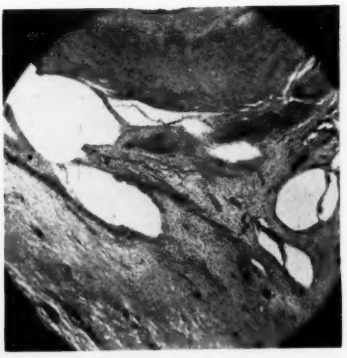


Fig. 3.

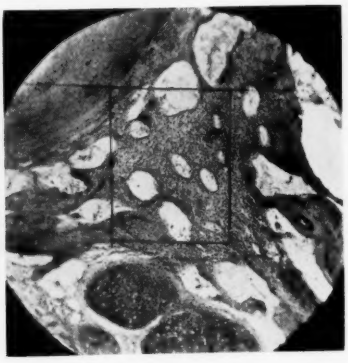


Fig. 2.

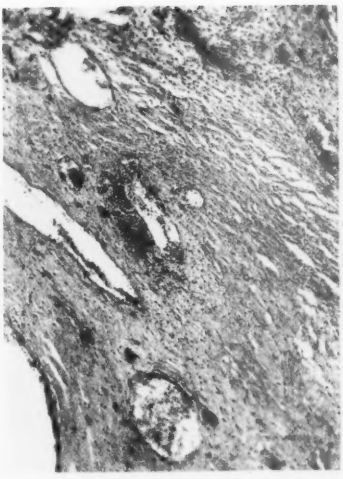


Fig. 5.

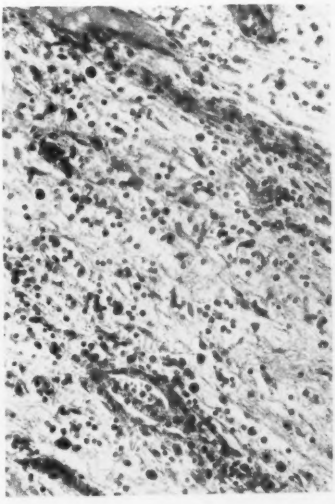


Fig. 7.

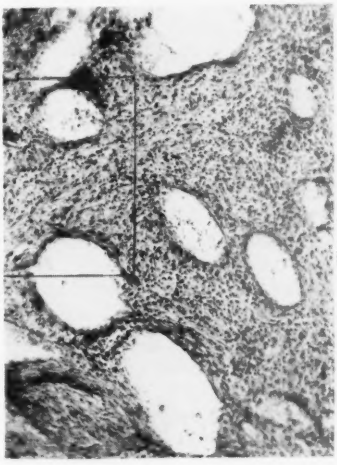


Fig. 4.

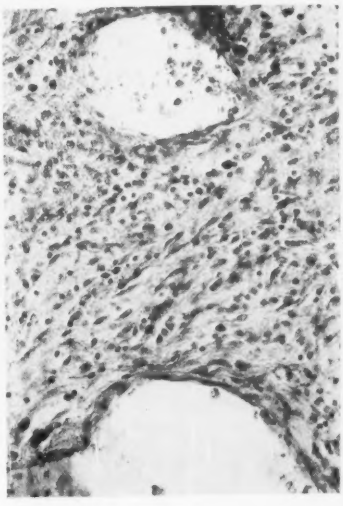


Fig. 6.

considerable daily chores. About eight or ten weeks before the end of school year a "boil" formed back of right ear; this discharged for two or three months, but at no time was attention directed in any way to the ear itself. During this time there was a discharging "boil" in the right inguinal region; occasionally headaches were admitted, rather than complained of.

Late in July, 1933, in the course of ordinary health, strength and usual physical activities, he put on heavy underwear, telling his father that he had been "feeling cold." For a week or two "sat around," "kind of tired," but soon "seemed all right again." At no time had headache been mentioned since before the end of spring school term. Without premonitory symptoms he awoke at 4 a. m., vomited three or four times, had several loose bowel movements, with restlessness but no pain. During the next five hours intermittently slept and vomited watery small amounts, gradually becoming disorientated and stupid; removed to hospital at 7 p. m., August 13th. Only cursory entrance examination was made by reason of his condition, "acute right tympanomastoiditis; apparently moribund." Death occurred within four hours.

Autopsy discovered immediate cause of death to be right tympanomastoid infection and brain abscess. Complete necropsy shed additional light upon the pathologic episode of 1930. The patient died with, and undoubtedly because of, right tympanomastoid infection, but by no means necessarily because of this alone; additional pathology revealed was too extensive to warrant concluding that the sole cause of death was "right mastoiditis with intracranial complications."

The following gross pathology was found:

Scalp.—Behind the right ear was found an irregular, wide scar such as might follow mastoid operation; surrounding skin showed some discoloration (careful inquiry established that no operation had been performed).

Skull.—On removal of the skull cap, dura was found adherent to the squamous portion of right temporal bone and adjacent brain; this adherent dura was set apart for subsequent microscopic study. Contiguous bone was softened and rough, with several irregular indentations containing yellowish fibrinous exudate; the groove between squamosa and petrosa was rough and pitted, apparently with vascular openings; bluish discoloration with yellowish flecks and some erosions extended medially into the petrosa and well toward the tip; temporal muscle was intimately adherent; above and slightly behind the beginning of the zygomatic process a fistula led to a large, irregular cavity; inner margin of anterior clinoid was definitely eroded with enlargement of entire sella such as occurs in obstruction of the fourth ventricle or the cerebral aqueduct; the hypophysis was compressed into a dished-out mass on the floor of the sella; the dura was adherent to the internal surface of the squamosa, its internal surface adherent to the temporal lobe. Nothing further of significance was found. Careful examination of cranial sinuses failed to reveal thrombus.

Brain.—Engorged surface vessels were found on the dorsolateral surfaces and bluish exudate covered the sulci, the pons and basilar cisterns including the cisterna magna, as if blue dye had been injected; the exudate was old and contained much fibrin.

Acute pathology was evident throughout right temporal lobe, which was larger than the left, with definitely flattened convolutions and squamosal adhesions. When the brain was removed yellowish gray pus exuded from the third ventricle

into which the brain abscess had ruptured; thromboses were found in the larger veins over the dorsolateral and basilar surfaces.

Section from right side with area of adherent dura through the internal capsule and tuber cinereum revealed encapsulated abscess within the right temporal lobe, 2.4 x 3.0 x 2.3 cm. Its wall consisted of irregular, brownish softened brain tissue, thin (yellow white) layer of connective tissue and heavy (greenish gray) layer of granulation tissue. From this capsule connective tissue pedicle 0.8 x 1.0 cm. extended laterally through the cortex of the second and third temporal convolutions. Contiguous brain tissue was yellowish, granular and disintegrating. The abscess had ruptured into the inferior horn of the ventricle, pus being found in the lateral ventricles. Lack of distortion of midline structures or ventricular contours suggest chronicity.

Section 1 x 5 cm. caudal to the one just described failed to reveal anything additional, but emphasized the almost cartilaginous nature of pedicle extending through the second and third convolutions to the adherent dura, and the presence of fibrinopurulent exudate within the lateral ventricle. On inspecting the anterior horn of the ventricle, the site of the abscess rupture was indicated by an accumulation of discolored exudate; volume of right cerebral hemisphere much greater than left.

Gross Findings.—Acute exacerbation of right chronic tympanomastoiditis, with squamosal osteomyelitis and fistula; dural adhesions to bone and to temporal cortex; encapsulated abscess of anterior right temporal lobe ruptured into inferior horn of lateral ventricle.

Further pathologic studies were made at the Los Angeles General Hospital, the University of Southern California and the University of Oregon. Drs. Frank Menne and Robert L. Benson, Dept. of Pathology, Univ. of Oregon School of Medicine, Portland, Oregon, report: "Microscopic examination of sections were made (see illustrations) through hypophysis, sella turcica, sphenoid sinus and dural investment—including internal carotid artery, sinus cavernosus, oculomotorius, trochlearis and ophthalmic division of trigeminus; dense fibrous connective tissue was found generally throughout left sinus cavernosus, with formation of new and irregular lumina lined with endothelium. These were numerous, often triangular or slit like; they contained blood as well as blood plasma. In the intervening fibrous connective tissue were diffuse accumulations of monocytes, histiocytes and occasional plasma cells (see Figs. 9, 10, 11 and 12), with a scattering of blood pigment granules within intercellular spaces and phagocytes; and miliary abscesses in which polymorphonuclear leucocytes predominate, with circumscribed collections of mononuclear phagocytes encircling discrete pyogenic foci. Under the oculomotor perineurium is an accumulation of polymorphonuclear leucocytes. This does not extend into the endoneurium, but merely elevates the perineurium. This inflamatory process extends to some extent in the direction of the plexus cavernosus where it assumes more chronic appearance. The dura extending over the hypophyseal surface reveals areas of abscess formation with ulceration. These small abscesses open upon the internal dural surface, on which masses of polymorphonuclear leucocytes and monocytes were found extending across to the opposite side, with here and there secondary smaller ulcers in the dura. To the right of the midline the purulent accumulation, consisting largely of polymorphonuclear leucocytes becomes more prominent; but the right sinus cavernosus reveals only moderate subacute perisinus innflammatory changes, not involving the lumina of the sinus.

Impression.—Old cavernous sinus thrombosis (left) with recanalizations; recent infection with miliary abscess production invading left oculomotor perineurium and extending upon the surface of adjacent dura. Because of limited number of sections examined, it is uncertain whether extension occurred to left side secondary to brain abscess of right side, or to latent pyogenic process in left sinus cavernosus (in course of exacerbation) breaking through and extending across to the right. There is no evidence of involvement of the hypophysis or of extension of infection into anterior lobe septa, sphenoidal sinus or floor of sella turcica. Sections made of this level, however, do not include posterior lobe of the hypophysis.

Pathologic Diagnosis.—Latent thrombotic cavernous sinusitis with recanalizations; recent miliary abscesses with ulcerations involving internal dural surface and left oculomotor perineurium, with slight extension to the left plexus cavernosus.

This case is unique in that it has afforded necropsy examinations of cavernous sinus tissues three years after clinical recovery from acute left infectious thrombosis. Additional studies of tissues concerned are in process and others are projected; examinations herewith reported reveal that the left cavernous sinus had undergone extensive thrombotic inflammation long before the onset of recent process characterized by "multiple miliary abscesses with ulceration." The pathologic nature of the fatal, acute process has been accorded especially searching inquiry by reason of possible relationship between it and the process of 1930 diagnosed "cavernous sinus thrombosis with recovery."

It is apparent the unsuspected pathology existed for a considerable period before culminating in right tympanomastoiditis, fistula, temporal lobe abscess, fibrinopurulent meningitis and death; the outcome of independent studies at the Los Angeles General Hospital, University of Southern California, and University of Oregon pathologic laboratories confirms the clinical diagnosis made in 1930—"Cavernous sinus thrombosis—recovery."

Thanks are especially expressed to Dr. Paul McKibben, Dean of Medical Department of University of Southern California, for fixing and sectioning this unusually difficult necropsy material; to Dr. Courville of Los Angeles General Hospital Resident Staff, for months of work required for decalcifying specimens; to Drs. Frank Menne and Robert Benson of the department of pathology, University of Oregon, for pathologic studies quoted in text; to Dr. Hall, of University of Southern California, department of pathology; to Drs. Jos. Reynolds and Herman Semenov of the Los Angeles General Hospital Resident Staff, Mr. Krajian of the Los Angeles General Hospital pathologic laboratory and Dr. Richard Millar of the California Scientific Photo Laboratory for much effective co-operation in the study of this unique case.

1154 ROOSEVELT BUILDING.

REFERENCE.

Lewis, E. R.: Cavernous Sinus Thrombosis: Recovery. ANNALS OF OTOLOGY, RHINOLOGY AND LARYNGOLOGY, 40:341 (June), 1931.

LXXXIX.

THE SURGERY OF THE GREAT SUPERFICIAL PETROSAL NERVE: ITS POSSIBLE RELATION TO SOME OF THE PATHOLOGY OF THE NASAL AND PARA-NASAL MUCOUS MEMBRANES.

EDWARD F. ZIEGELMAN, M. D.,

SAN FRANCISCO.

The pioneer work of Cushing,[1] Frazier, Royle,[2] Adson and his co-workers[3] on the cerebrospinal, sympathetic and parasympathetic nervous systems is well known. Langley,[4] Kuntz[5] and others have contributed to our knowledge of the finer anatomy and physiology of this problem. The postoperative results in scleroderma, Raynaud's, Buerger's and in Hirschsprung's disease, by section of a portion of the lumbar sympathetic has stimulated further investigation.

Though the sympathetic, parasympathetic and cerebrospinal nervous systems are very closely related to the ear, nose and throat, this relationship (especially the anatomic) is extremely difficult to utilize surgically. The observations of the pathology have been bacteriologic rather than neurologic. By contrast the lumbar sympathetic plexus and ganglia are relatively simple, likewise the stellate ganglion in the field of the thoracic or general surgeon. It is evident the nearer one gets to the central nervous system, the more difficult becomes the surgical approach to the various plexuses, ganglia and nerve fibers related to the sympathetic and parasympathetic. A few bold pioneers have blazed the trail. A small number of them have been otolaryngologists. Sluder[6] probably did most to stimulate members of the specialty to realize the importance of the relationship of nervous disturbances. Fenton and Larsell[7] have attempted to place the embryology and histology of some of the nerves related to the sphenopalatine ganglion on a sound scientific basis. Ruskin[8] has shown the relationship of neurogenic disturbances to clinical nasal pathology. Abroad Müller,[9] in his excellent text, has added new light. Many years ago the researches of Jonnesco[10] stimulated great activity in the field of the sympathetics and their relation to clinical surgery.[11]

*Read before the twenty-second annual meeting of the Pacific Coast Oto-Ophthalmological Society, Butte, Mont., July 16-19, 1934.

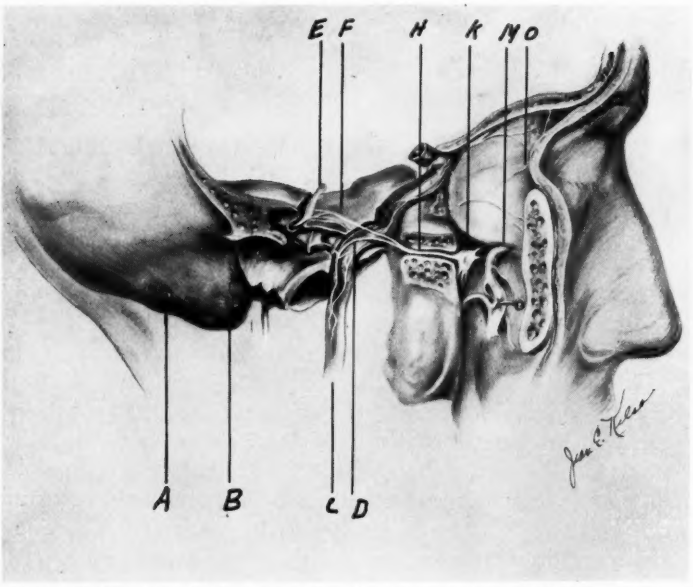


Fig. 1. Photograph of drawing from a dissection by the author, modified by the artist. It demonstrates at *A* the mastoid foramen, *B* the mastoid process, *C* the internal carotid artery, *D* the sympathetic plexus about the artery which joins the great superficial petrosal nerve *F* to form the vidian at *H*. *E* the facial nerve with geniculate ganglion from which the great superficial petrosal nerve is given off. The sphenopalatine ganglion at *K*, *M* the superior maxillary nerve dissected free, *O* the orbit.

Cathcart and Benedict[12] believe that but 25 per cent of human energy is under the control of the will and that the other 75 per cent is subconscious. This statement may seem theoretical and philosophic, but we may recall the saying of Huxley, quoted by W. J. Mayo:[12] "He that does not go beyond the facts will seldom see the facts." Opinions such as these have caused the pendulum of surgery to swing from that of gross pathology to the study of the histopathology, physiology, physics and chemistry of the cell and its structures.

Since Rosenow's masterly work on focal infections,[13] the oto-rhino-laryngologist has been closely allied to the observations that he advanced, chiefly through the field of sinus and tonsillar infection. There is still disagreement in the profession as to their being a source of infection, as well as to treatment.[14-20] The opinion of many that the sinus mucosa is a nidus for systemic invasion has brought about a great deal of observation and study on the histology and pathology of these membranes.[21-23] For years Lewis has been advocating a further search as to etiology.[24]

The nasal and paranasal mucous membranes are closely related to the nervous system, both the cerebrospinal and the vegetative.

Again I must refer to Langley,[4] who states that the so-called parasympathetic fibers come from the bulbar portion of the brain stem. They are carried by way of the third, seventh, ninth, tenth and eleventh cranial nerves to the various ganglia and structures about the head and neck. He also states that the parasympathetic fibers connect with the fifth nerve. To quote Harkness:[25] "The mucous membranes of the nose, accessory sinuses, buccal cavity and pharynx receive a double involuntary innervation, the fibers following those of the fifth nerve from the sphenopalatine ganglion, the sympathetic originating through the great deep petrosal nerve and the parasympathetic through the seventh nerve." If opinions such as these be correct, then it is self-evident that one of the pathways of parasympathetic innervation to the sphenopalatine ganglion must be by way of the great superficial petrosal nerve.

The anatomy of the sphenopalatine ganglion is that of the second division of the fifth cranial nerve.[26] It lies in the pterygomaxillary fossa and is triangular in shape. It is situated just below the superior maxillary nerve, more or less in a condition of suspension from the main trunk as it passes through the fossa to enter the infraorbital canal. It possesses a motor, a sensory, a sympathetic and a parasympathetic root.[27] Its sensory root is derived from the superior maxillary, its motor and parasympathetic from the facial, directly and by way of the intermediate nerve of Wrisberg, and its sympathetic from the carotid plexus. The parasympathetic, through the great superficial petrosal and the sympathetic through the great deep petrosal enter the posterior portion of the ganglion as the vidian nerve (Fig. 1). Delie's hypothesis that the vidian nerve includes fibers of vagus origin, which join the superior cervical sympathetic ganglion and continue upward in the internal carotid nerve has apparently been confirmed by Kuntz.[28] The branches of distribution of the sphenopalatine ganglion are divided into four groups: ascending to the orbit, descending to the palate, internal to the nose and posterior to the nasopharynx (Fig. 2). The ganglion serves as a synapse between the preganglionic and postganglionic fibers.

In all methods of approach applied to the ganglion, either by injection or for removal, all the fibers and ganglion cells are involved.[6 27 29 30] For anesthesia this is desirable.[31 32] No attempt has been made to isolate the respective tracts as far as therapy is concerned. If the lesion of the nasal mucosa was one of dilation, then to overcome it by any of the methods advocated it was necessary to destroy the vasoconstricting fibers, also the motor, sensory and dilating as well. Therefore in attempting to overcome one pathologic

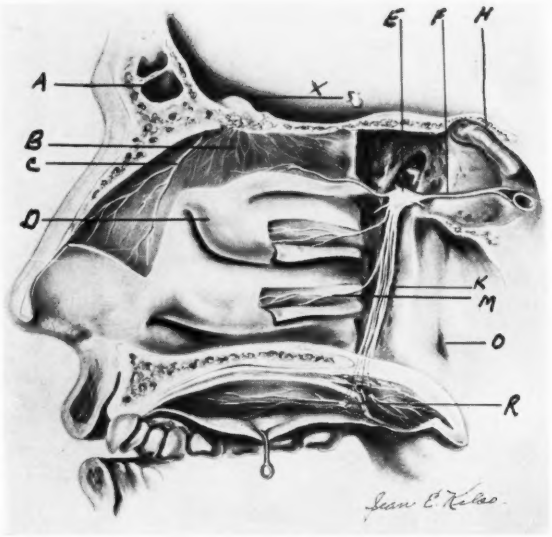


Fig. 2. Photograph of a drawing from a dissection by the author, modified by the artist. It demonstrates at *A* the frontal sinus, *B* the olfactory nerves, *C* the anterior nasal nerve, *D* anterior tip of middle turbinate, *E* sphenopalatine ganglion, *F* the vidian, *H* the carotid, *K* posterior palatine nerves, *M* branch to turbinate, *O* the eustachian orifice, *R* branches of distribution of descending palatine nerves in hard and soft palate.

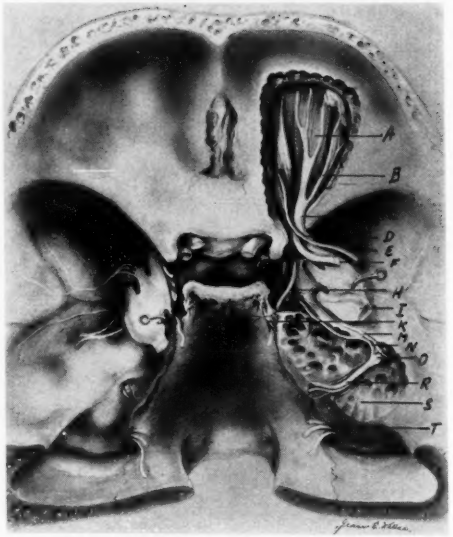


Fig. 3. Photograph of a dissection by the author. *A* orbit, *B* lacrymal nerve, *C* ophthalmic branch of trigeminal, *D* third nerve, *E* the middle fossa, *F* fourth nerve, *H* sixth nerve, *I* gasserian ganglion, with motor root showing on under surface of everted ganglion, *K* petrosphenoid ligament with sixth nerve passing beneath in Dorello's canal, *M* the small superficial petrosal nerve, *N* the great superficial petrosal nerve, *O* geniculate ganglion, *R* the internal auditory canal uncapped, showing the facial, auditory and intermedius, *S* the petrosal pyramid, *T* ninth, tenth and eleventh nerves.

lesion another was formed, or at least the anatomic structures were destroyed or inhibited. This may have accounted for failures in the treatment of various lesions of the nose by way of the sphenopalatine ganglion.

From clinical observations on head injuries, I am convinced that a marked turgescence of the nasal mucosa, especially over the turbinates, is frequently present. This is similar to conditions occurring in the internal ear.[33] It is my opinion that much of the vasodilating fibers to the nose and paranasal mucosa come from the brain by way of the parasympathetic through certain bundles in the facial nerve, probably by way of the intermediate nerve of Wrisberg (Fig. 3). This hypothesis and conclusion, based upon a review of the literature,[34] may not seem so theoretical when we realize that the vasodilating fibers to the submaxillary gland are through the chorda tympani.[35] As we know, this nerve comes through bundles in the facial from the brain. In contradistinction, the great deep petrosal nerve carries vasoconstricting fibers to the sphenopalatine ganglion. To summarize: The great superficial petrosal nerve regulates all or part of the vasodilating, and the great deep petrosal all or part of the vasoconstricting activities to the nasal and paranasal mucosa. The vidian nerve contains both efferent and afferent fibers. Therefore it is apparent that impulses both centrally and peripherally must pass through this nerve and its two main nerves of formation, the great superficial and the great deep petrosal. Any treatment applied to these nerves must take into consideration these facts. Whether one believes that pathology of the nasal and paranasal mucous membranes is a local cause for systemic disturbances, or a local manifestation of a systemic cause, the applicability of treatment to the nervous pathways is similar. Conceding that a neurogenic transmission is possible, then treatment applied to the nerve or nerves would appear sound. Vail,[36] under the description of "great superficial petrosal nerve neuralgia," mentions a type of pain.

Working upon facts determined from the literature, in addition to personal clinical observations, I am presenting a surgical method for the treatment of some of the neurogenic disturbances of the nasal and paranasal mucous membranes. Fundamentally the principle depends upon the anatomic fact that the great superficial petrosal nerve, which apparently is to a great degree the vasodilator nerve to the sphenopalatine ganglion, after turning medially from the internal auditory canal, in very close relation with, though not an integral part of the geniculate ganglion,[37] travels for a very limited distance subperiosteally and then becomes extradural. It leaves the bone of

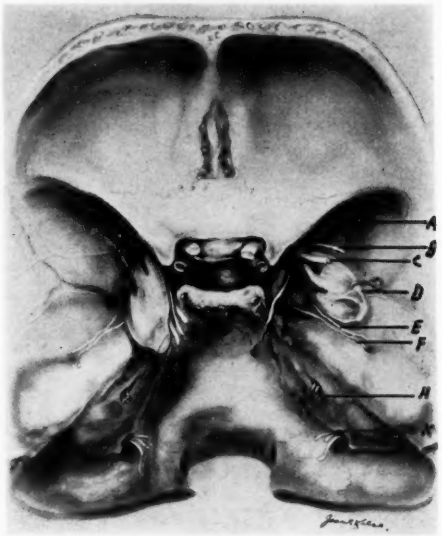


Fig. 4. Photograph of a drawing from a dissection by the author. *A* middle fossa, *B* third nerve retracted, *C* fourth nerve, *D* gasserian ganglion, *E* small superficial petrosal nerve, *F* the great superficial petrosal nerve, *H* the internal auditory meatus with seventh, eighth and intermedius nerve, *K* ninth, tenth and eleventh nerves, *L* the sixth nerve displaced onto superficial surface of petrosphenoid ligament. Normally it passes beneath this ligament through Dorello's canal, *M* hypoglossal nerve passing through anterior condyloid foramen.

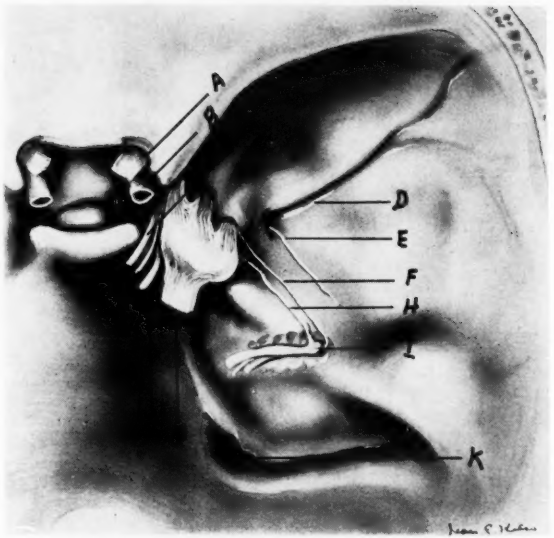


Fig. 5. Photograph of drawing from a dissection by the author. *A* the optic nerve, *B* internal carotid artery, *C* third nerve, *D* foramen spinosum and middle meningeal artery, *E* external petrosal nerve, *F* small superficial petrosal nerve, *H* the great superficial petrosal nerve, *I* geniculate ganglion, also uncapped internal auditory canal showing nerves passing through it, *K* jugular foramen, *M* fourth nerve, *N* sixth nerve, *O* sensory root of gasserian ganglion from anterior aspect may be seen the three branches of distribution.

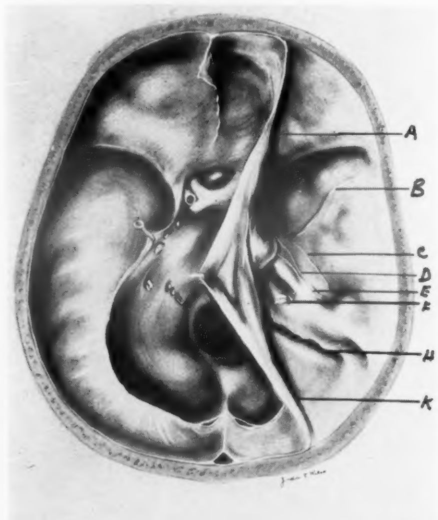


Fig. 6. Photograph of a drawing from a dissection by the author. *A* anterior fossa. *B* middle meningeal artery. *C* small external petrosal nerve, to foramen spinosum. *D* small superficial petrosal nerve. *E* great superficial petrosal nerve. *F* internal auditory meatus showing seventh, eighth and intermedius nerves. *H* depression for lateral sinus. *K* retracted dura.

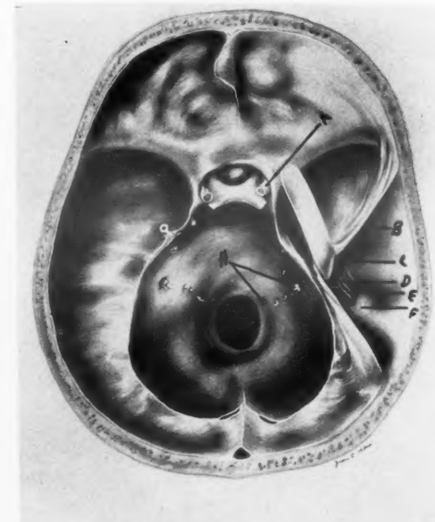


Fig. 7. Photograph of a drawing from a dissection by the author. *A* internal carotid artery, optic chiasm and sella. *B* retracted dura. *C* external petrosal nerve. *D* small superficial petrosal nerve. *E* great superficial petrosal nerve. *F* eminentia arcuata. *H* seventh, eighth, ninth, tenth, eleventh and twelfth cranial nerves passing through meninges to respective foramen of exit.

the anterior surface of the petrosal pyramid usually just internal to the eminentia arcuata by way of the hiatus fallopii, somewhat external to the foramen spinosum, through which passes the middle meningeal artery and the small external petrosal nerve. The eminentia arcuata is the anatomic landmark. The structures to avoid in isolating the nerve are the middle meningeal artery and the small superficial petrosal nerve. It lies superior and superficial to this nerve, after it leaves the hiatus fallopii, and from which it may be isolated anatomically and surgically as both nerves pass medially, the one to enter into the formation of the vidian, the other to pass through the foramen ovale to join the otic ganglion. (Figs. 4, 5, 6, 7.)

The great superficial nerve lies in a position for surgical treatment via a subtemporal approach, as is utilized in gasserian ganglion surgery, or as has been more recently advised for petrosal tip suppuration.[38] The nerve may be isolated by way of the tegmen tympani, exposing the geniculate ganglion and severing it just internal to this ganglion. This method is much more difficult and in addition is accompanied with great hazard to the facial nerve, as well as to the small superficial petrosal. The chief requisite in a satisfactory result is the utilization of the electric spatula advocated by Frazier in his approach to the Gasserian ganglion. In addition, one must be well versed in the anatomy of the petrosal pyramid and its adjacent fossa and possess a fundamental surgical training. (Figs. 8, 9.)

What pathologic lesions this method may be applicable to I am unable to state. Much of this presentation is based upon assumptions derived from a survey of the literature and a limited amount of clinical observations in head injuries with concomittant nasal pathology during the past quarter of a century. A study of the surgical anatomy of this nerve stimulates an imaginative mind to a vista of many possibilities. This is referable to pathology central, peripheral or along its course in relation to petrosal pyramid lesions.

Its virtue will depend in a great measure upon the physiologist and his ability to prove or disprove the neurogenic manifestations in the nose and their relation to the great superficial petrosal nerve. To quote Crile: "The final analysis of all scientific medicine is the crucible of the clinic."

CONCLUSIONS.

1. From clinical observations the great superficial petrosal nerve carries impulses of a vasodilating character to the nasal and paranasal sinus mucosa.

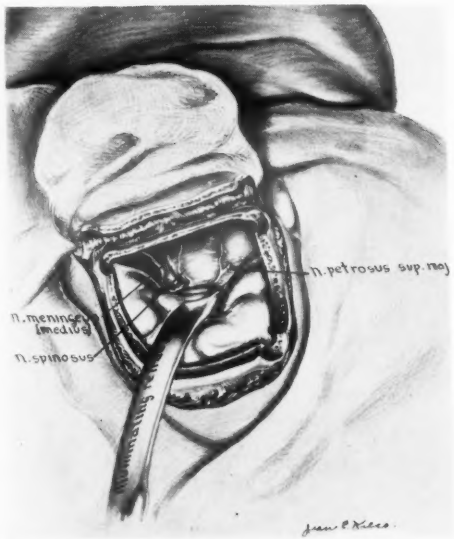


Fig. 8. Photograph of a drawing showing the osteoplastic flap retracted. It demonstrates the great superficial petrosal nerve as it becomes extra dural, just medial to the eminentia arcuata. The Cushing muscle splitting approach is preferable to this osteoplastic in the actual surgical approach.

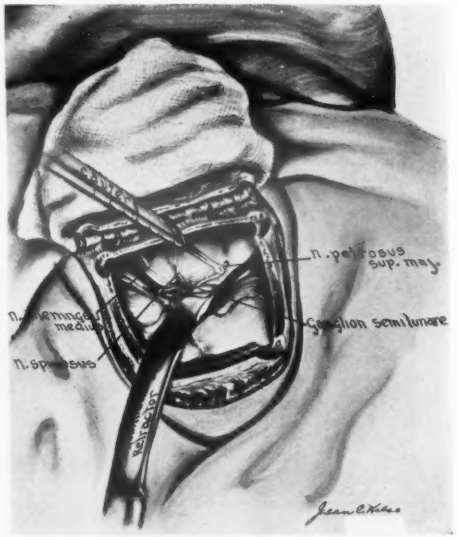


Fig. 9. Photograph of drawing to demonstrate the technic and method of severing the great superficial petrosal nerve.

2. From the literature this opinion is corroborated.

3. Many of the pathologic lesions of the nose and accessory structures are probably due to a central impulse carried by way of the great superficial petrosal nerve.

4. Many of these lesions may be classified at present in the category of allergy, rhinitis, polyposis, etc.

5. From another point of view this nerve probably serves as a pathway for reflex transmission, locally or systemically, of vague pains and neuralgias, from nasal and paranasal pathology.

6. If such should prove to be true, they may be treated by the method described.

7. Its virtue will depend upon the increasing physiologic knowledge of the structures involved.

450 CENTER ST.

BIBLIOGRAPHY.

1. Cushing, Harvey W.: "Tumors of the Nervus Acusticus and the Syndrome of the Cerebellopontile Angle. Phila., W. B. Saunders, 1917.

2. Royle, N. D.: "Surgical Treatment of Raynaud's Disease and Similar Conditions. M. J. Australia, 2:341, 1927.

3. Adson, A. W.: "Results of Sympathectomy in Treatment of Raynaud's and Buerger's Disease." Proc. Interstate Postgrad. M. Assemb. N. A., 3:386, 1928.

Adson, A. W., and Masson, James C.: "Dysmenorrhea Relieved by Resection of Presacral Sympathetic Nerves." J. A. M. A., 102:986-990, March 31, 1934.

Adson, A. W., and Brown, G. E.: "Malignant Hypertension." J. A. M. A., 102:1115-1118, April 7, 1934.

4. Sharpey-Schäfer, E. A.: "Textbook of Physiology." London, Pentland, 1898-1900, v. 2.

Langley, J. N.: Das sympathische und verwandte nervöse Systeme der Wirbeltiere (autonomes nervöses System). Ergeb. d. Physiol., v. 2, pt. 2, pp. 818-872, 1903.

Langley, J. N.: "The Autonomic Nervous System." Brain, 26:1-26, 1903.

Langley, J. N.: "The Nomenclature of the Sympathetic and of the Related Systems of Nerves." Zentralbl. f. Physiol., 27:149-152, 1913.

5. Kuntz, Albert: "The Autonomic Nervous System," Phila., Lea & Febiger. 1929.

6. Sluder, G.: "Concerning Some Headaches and Eye Disorders of Nasal Origin." St. Louis, C. V. Mosby Co., 1918.

7. Fenton, Ralph A., and Larsell, O.: "The Embryology and Neurohistology of the Sphenopalatine Ganglion Connections." Trans. Am. Otol. Soc., 18:183, 1928.

8. Ruskin, S. L.: "The Neurologic Aspects of Nasal Sinus Infections." Arch. Otolaryng., 10:337, Oct., 1929.

9. Müller, L. R.: "Die Lebensnerven." Berlin, J. Springer, 1924.

10. Jonnesco, T.: "Le sympathique cervico-thoracique." Paris, Masson, 1923.

11. Mayo, C. H.: "Collected Papers of the Mayo Clinic, 1905-09." Phila., Saunders, 1911, pg. 438.

12. Cathcart and Benedict, quoted by Mayo, W. J.: "Co-ordination of Human Vegetative Functions." Surg. Gynec. and Obst., 38:312-317, March, 1924.

13. Rosenow, Edward C.: "Elective Localization of Streptococci." Coll. Papers of the Mayo Clinic, 7:764-778, 1915.

14. Anderson, Carl M.: "Suppuration in the Paranasal Sinuses as a Factor in Focal Infection: Review of 400 Cases." Coll. Papers of the Mayo Clinic, 22:594-598, 1930. Also: J. A. M. A., 94:1889-1891, 1930.

15. McGinnis, Edwin: "Nonsuppurative Ethmoiditis." Trans. Am. Laryng. Soc., 52:141-147, 1930.

16. Halle, Max: "Intranasal Operation on Frontal Sinus." In:

Loeb, Hanau: "Operative Surgery of Nose, Throat and Ear." St. Louis, C. V. Mosby, 1916-1917.

17. Moure, Jean Gabriel Emile: "Guide pratique des maladies de la gorge, du larynx." Paris, O. Doin, 1908.

18. Faulkner, E. R.: "Discussion of Paper by McGinnis." Trans. Am. Laryng. Soc., 52:145-146, 1930.

19. Mosher, Harris P.: "Intranasal Operation on Frontal Sinus." See reference No. 16.

20. Sewall, E. C.: "External Operation on Ethmosphenoid Frontal Group of Sinuses Under Local Anesthesia." Arch. Otolaryng., 4:377-411, Nov., 1926.

21. Lynch, R. C.: "Symposium on Ethmoidal Sinusitis: External Radical Approach." ANNALS OF OTOLOGY, RHINOLOGY AND LARYNGOLOGY, 38:931-939, 1929.

22. Gorham, C. B., and Bacher, J. A.: "Regeneration of the Human Maxillary Antral Lining." Arch. Otolaryng., 11:763-771, June, 1930.

23. Kistner, Frank B.: "Histopathology and Bacteriology of Sinusitis with Comments on Postoperative Repair." Arch. Otolaryng., 13:225-237, Feb., 1931.

Kistner, Frank B.: "Nasal Sinus Infection Associated with Diseases of the Vascular System." ANNALS OF OTOLOGY, RHINOLOGY AND LARYNGOLOGY, 43:88-93, March, 1934.

24. Lewis, Eugene: "Significance of Hydrops Mucosæ." Trans. West. Sect. Am. Laryng., Rhin. and Otol. Soc., Seattle, 1931, v. 37:420-425.

25. Harkness, G. F.: "The Involuntary Nervous System in Its Relation to Otolaryngology." Trans. A. M. A. Sect. Laryng., Otol. and Rhin., 1933, pp. 85-107.

26. Vogel, Klaus: "Die Beziehungen des Ganglion Sphenopalatinum zu den von ihm ausgehenden Nervenstämen. Ztschr. f. Hals-Nasen- u. Ohrenheilk., 25:485,502, March. 1930

27. Ruskin, S. L.: "Contributions to the Study of the Sphenopalatine Ganglion." Laryngoscope, 35:87-108, 1925.

28. Kuntz, Albert: "Nerve Fibers of Spinal and Vagus Origin Associated with the Cephalic Sympathetic Nerves." ANNALS OF OTOLOGY, RHINOLOGY AND LARYNGOLOGY, 43:50-66, March, 1934.

29. Sewall, E. C.: "An Operation for the Removal of the Sphenopalatine Ganglion." ANNALS OF OTOLOGY, RHINOLOGY AND LARYNGOLOGY, 35:1-22, 1926.

30. Vogel, Klaus: "Histopathologische Befunde am Ganglion Sphenopalatinum mit besonderer Berucksichtigung der Atrophischen Rhinitiden." Ztschr. f. Hals-Nasen- u. Ohrenheilk., 22:507-553, Feb., 1929.

31. Ziegelman, Edward F.: "The Inverted T-Shaped Incision with Block Anesthesia in Radical Surgery of the Maxillary Sinus." West. J. Surg., Obst. and Gynec., 42:103-109, Feb., 1934.

32. Braun, Heinrich: "Local Anesthesia." Phila., Lea & Febiger, 1914, p. 216.

33. Grove, W. E.: "The Ear in Head Injuries: Practical Considerations." ANNALS OF OTOLOGY, RHINOLOGY AND LARYNGOLOGY, 40:222-241, March, 1931.

34. Wachsberger, Alfred: "Rôle of the Sympathicus and Parasympathicus in the Etiology of Atrophic Rhinitis." Arch. Otolaryng., 13:265-269, Feb., 1931.

35. Howell, W. H.: "Textbook of Physiology." Phila., Saunders, 1926, p. 618.

36. Vail, Harris H.: "Syndrome of Pain in Its Reference to the Eye, Ear, Nose and Throat." Trans. Am. Acad. Ophth. and Otolaryng., 38th annual meeting, p. 255-264, 1933.

37. Lenhossék, Joseph von: *In*—Müller, L. R.: "Die Lebensnerven." Berlin, J. Springer, 1924, p. 129.

38. Unterberger, S.: "Anatomisches zur Streitschen Pyramidenspitzenfreilegung bei Gradenigoschem Symptomenkomplex nebst Mitteilung eines so operierten Falles." Ztschr. f. Hal.- Nasen- u. Ohrenheilk., 34:293-301, 1933.

XC.

AUDITORY FATIGUE INCLUDING A NEW THEORY OF HEARING BASED ON EXPERIMENTAL FINDINGS.*

E. M. JOSEPHSON, M. D.,

NEW YORK.

This is a preliminary report of paradoxical results obtained in a series of experiments undertaken to study fatigue of hearing, with special reference to the part played by the auditory end organ. No effort has been made in work on the subject hitherto reported to differentiate the relative involvement in fatigue of hearing of the various factors entering into play—sensation, perception, judgment and others—or to differentiate the rôle played by the various parts of the highly complex end organ, the ear.

Much has been assumed with regard to auditory fatigue which is not justified by the data at hand. Fatigue of hearing obviously accompanies sleep. But even most casual observation indicates that, of all the senses, hearing is most alert in sleep. Dr. Donald A. Laird has stated in his volume[1] "Sleep" that "in going to sleep . . . . ability to hear noises is lost last . . . and recovered first." In other words, the ears are the most alert sentinels of the body. Studies on auditory memory during sleep and its effect on the learning process in the waking state make it appear extremely doubtful that it is correct to assume that even in bodily fatigue and sleep is there fatigue of the auditory end organ. No hearing curves for the sleeping state are as yet available. Action currents obtained from the auditory nerve of the anesthetized animal give evidence of need for further study and research in these directions. It might be noted, however, that Dr. Hallowell Davis has found that the depth of anesthesia does affect the limit of auditory nerve action current frequency; the limit of waves of amplitude fully equal to that of the stimulating frequency was 700 cycles under deep anesthesia, and 1,000 cycles under light anesthesia. The full effect of anesthesia upon nerve action currents in the auditory tracts has not yet been determined, though it must obviously completely inhibit the action of the higher centers when it reaches a certain depth.

*Read before the Section on Psychology, American Association for the Advancement of Science, Harvard University, Cambridge, Mass., December 28, 1933.

The physiology classroom experiment in which one ear is subjected to stimulation with a note for a prolonged period, and the loudness of the same note for the unstimulated ear was found to be higher, is by many observers regarded as conclusive evidence of fatigue of the ear. Refinements of this experiment were made by a number of observers, including von Bekesy,[2] and MacDonald[3] and Allen, who carefully studied the differential intensity before and after more or less prolonged stimulation with moderate intensities of a frequency, and found an increased differential which returned, after rest, to a figure higher than normal and then to normal in a period of fifteen minutes. But the judgment of intensity is a mental process involving many factors other than mere sensation, and the findings cannot be regarded as evidence of fatigue of the end organ.

Casting about for a clinical means of testing fatigue of the ear, it was resolved that changes in the threshold of intensity would more truly give a measure of fatigue changes in the end organ. For though many processes are involved in the hearing of a note, at threshold the entire chain of them is critically bound up with sensation and the condition of the end organ.

The method of observation was as follows: A phone was fixed in position over the ear throughout the experiment by means of a headband, in order to avoid errors introduced by changing the position of the phone over the ear. The ear was stimulated with a single frequency generated by a beat-frequency oscillator, and the threshold determined. After a lapse of ten minutes, the ear was continuously stimulated with the same frequency at higher intensity levels. At set times the threshold was redetermined. The attenuator permitted of measurement in terms of steps of three decibels. Intensities higher than half the difference between threshold and feeling level of the frequency were not used for fatiguing, because they were found to give rise to confusing tinnitus.

In the normal ear it was found that no fatigue followed prolonged stimulation, provided that environmental conditions, such as barometric pressure, remained constant. On the contrary, it was found that lesser intensities became audible, that the threshold had been lowered. A result diametrically the opposite of fatigue was found.

In subjects suffering from progressive deafness as well as in subjects suffering from various degrees of cardiac decompensation, in both of which types there is an impairment of the circulation of the ear, there was found, on the other hand, marked and rapidly pro-

gressive rise of threshold, and fatigue on prolonged stimulation. This fatigue is often so rapid that in the course of one minute the threshold may rise as much as fifteen decibels, and may lead the observer to suspect that the subject is unreliable and has really not heard the lower threshold intensity. In these cases, excepting the most advanced types of deafness, the hearing becomes more or less stabilized at a higher level than the initial threshold. Even at this higher threshold level, however, it is found that the hearing is intermittent in the course of continuous stimulation. In these periods of intermittency, which vary in duration from seconds to minutes, it is found that there has been further fatigue, that the threshold has risen as much as six or more decibels. But in the course of continuous stimulation there is recovery to the stabilized threshold level, in contrast to failure of recovery to the initial, transient threshold levels. Recovery to the initial, transient threshold level does not occur during the period of stimulation but only after a prolonged period of rest.

This fatigue of hearing manifested by a rising threshold, I have found to be one of the earliest diagnostic signs of progressive deafness. It is elicitable before any material loss of hearing takes place. It should play a rôle in the early diagnosis of progressive deafness and in preventive therapy.

In the study of the relation of vascularity of the ear to this phenomenon of fatigue, it was found that such fatigue manifested by a rise in threshold could be induced in normal subjects by pressure on the carotid artery on the same side. It was also found that when the barometer was dropping in the course of experiments on normal subjects there was no drop in the threshold after prolonged stimulation; instead, the threshold remained stationary in most subjects, or rose fractionally in some. This effect of barometric pressure on the hearing manifests itself in cases of progressive deafness, as previously reported, by a very sharp rise in hearing threshold. The interpretation offered for the effect of changes in barometric pressure is that it influences vascularity of the ear and head by reason of diminished air pressure on the body surface. The influence of vascularity on the threshold bears out earlier observations made by Wever and Bray[5] in the cat, and by Dr. A. A. Gray and myself[6] in the human, that interference with the blood supply of the ear results in impairment of hearing, which is repaired by restoration of the blood supply.

The absence of fatigue in the ear of the cat has been noted recently by Drs. Davis, Saul, Lurie and Derbyshire in the course of their work on cochlear spread currents. With the exception of an initial surge in the initial cycles, which might be interpreted as com-

parable to initial current surge in alternating current circuits, they informed me that they have found no change in the amplitude of oscillograph curves of cochlear spread when the stimulating frequency was maintained at constant intensity over prolonged periods of time. This might possibly be regarded as experimental proof of the absence of fatigue in the cochlea.

Some data on the origin of the paradoxical phenomenon of increased sensitivity of the ear after prolonged stimulation is offered by further studies. In the normal ear it was found that the rate of increase of sensitivity, or drop in threshold, increased as the intensity of the stimulating note increased, within the limit of feeling level.

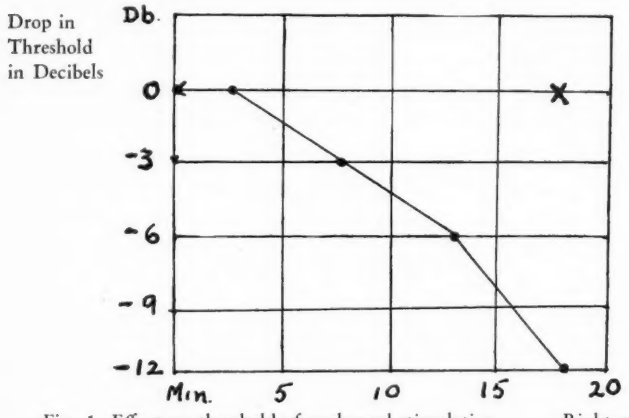


Fig. 1. Effect on threshold of prolonged stimulation. . . . .Right ear—normal, tympanic membrane intact. xx Left ear—large perforation in tympanic membrane; threshold 15 db higher than right. 0 represents initial thresholds. Frequency 1000 cycles. Intensity of stimulation for the first 15 minutes was plus 30 db and for the final 5 minutes 60 db above threshold. The right ear showed a drop in threshold which was accelerated when the stimulating tone was doubled in intensity. Left ear showed neither drop nor rise in threshold.

Stimulation with subliminal intensities over prolonged periods of time did not result in bringing those intensities within the range of audibility. This finding led to a study of the fatiguing effects of prolonged stimulation upon ears the tympanic membranes and ossicles of which had been destroyed by disease process. I selected from a large series of these cases which have been freed of infection with recovery of hearing to levels within the range of normal, a number of subjects in whom only one ear had been involved and the other was normal. In the normal ear there was found a lowering of the threshold after prolonged stimulation; whereas, in the ear from which the ear drum and ossicles were missing, the threshold remained constant

throughout the period of stimulation. Fig. 1 is the graph of the findings in one such case, which is typical of the group. The data indicates that the auditory accommodative mechanism of the middle ear, comprised of the tympanic membrane, the ossicles, and the stapedius and tensor tympani muscles, gives rise to the paradoxical reaction of increased auditory sensitivity following prolonged stimulation. The action of this mechanism in response to prolonged stimulation is attested to by the sensation of tension in the ear which supervenes in the course of stimulation and persists for a fairly long period thereafter.

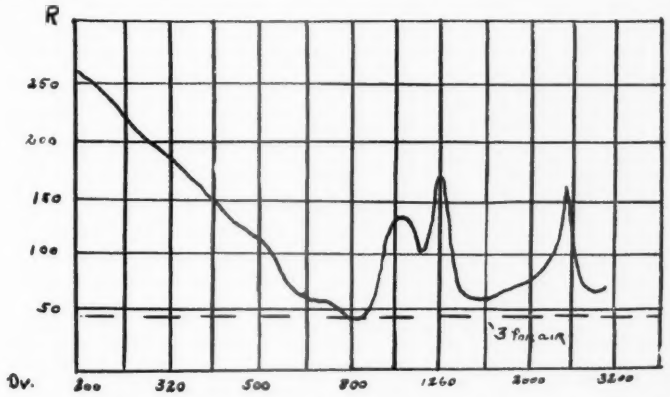


Fig. 2. Apparent Impedance of the Tympanic Membrane.—J. Troger, Physikalische Zeitschrift, XXXI.

The calculated resistance of the tympanic membrane, which does not include the element of phase, is charted in terms of c. g. s. units. The value for 800 dv matches that for air.

Some light is thrown on the nature of this mechanism by the work of Johann Troger[7] on the measurement of the impedance offered to sound by the tympanic membrane and the auditory accommodative mechanism, by means of standing waves. He showed that this resistance varies with frequency; that it is highest for low frequencies, dropping steadily to a minimum at 800 cycles, varying irregularly for higher frequencies. (Fig. 2). Further data in this direction should be sought.

Clinically it has been shown that impedance of the tympanic membrane is varied by the auditory accommodative mechanism. It has also been demonstrated by me that alterations in the structure of the tympanic membrane, labeled "interstitial myringitis," cause a heightened impedance to sound and are an element in the deafness of progressive deafness; and that this impedance may be reduced for

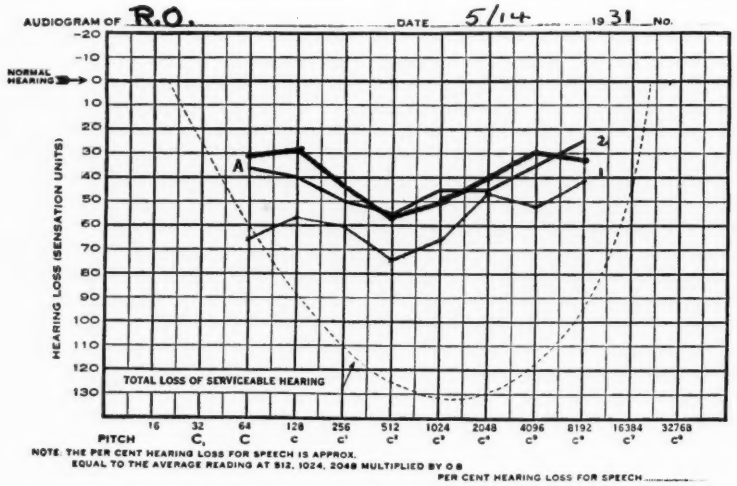
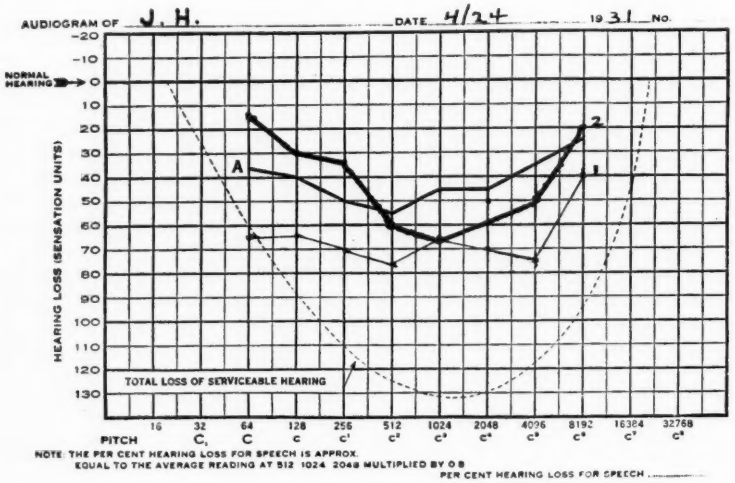


Fig. 3. Audiogram illustrating the effect of incision of the tympanic membrane on the threshold of hearing in progressive deafness cases. *1* is curve of threshold before incision. *2* is curve after incision. *A* is bone conduction curve.



selected frequencies by properly placed incisions in the tympanic membrane. (Fig. 3.) The pathologic changes reported by many observers in the muscles of the accommodative mechanism of the middle ear in progressive deafness and the above stated factors will undoubtedly be found to enter as factors in the fatigue reactions of progressive deafness.

Auditory fatigue cannot be explained in terms of fatigue of the auditory nerve end organs, in the normal. The origin of auditory fatigue must be sought in other parts of the auditory mechanism—in the elements of perception, apperception, attention, judgment and others involved in the hearing process. A grave obstacle in the way of study of the cerebral elements of fatigue of hearing is the failure of definite localization of the higher centers of hearing in the cortex. Pavlov[8] and his collaborators have demonstrated in the dog total and complete deafness can be produced only by bilateral decorticization of the cerebrum "posterior to a line starting from a point above and immediately behind the gyrus sigmoideus, stretching to the tip of the gyrus sylviaticus and then passing along the fissura fossæ sylvii." Measures less extensive, or limited to the temporal lobes, did not abolish sound conditioned reflexes. Lesions of the colliculi are known, clinically, to cause complete and bilateral deafness. But whether or not there exist higher or cortical centers of hearing has not yet been determined. The following case, showing a bilateral, symmetrical and symmetrically varying island of deafness, makes it appear that such a center is possible.

REPORT OF A CASE.

Case 1.—J. G. Male. Age 36 years. C. C. Deafness—Duration ten years. Past history—Measles and diphtheria in childhood; influenza, 1927; tonsillectomy and adenectomy because of infrequent colds, 1928. History—Deafness, more marked in left ear. No tinnitus or vertigo. Occasional severe headaches. Otherwise negative.

Physical examination—Cupping of disc absent in left eyeground, and kinking of vessels of retina marked, contrasting with fairly normal right eyeground. Knee jerk slightly exaggerated on right side. Otherwise negative.

Ear examination—Tympanic membranes and external auditory canals appear normal. Bone conduction more prolonged than air conduction. Bilateral and symmetrical island of deafness which varies in extent symmetrically from time to time. See chart, Fig. 4.

The findings of absence of fatigue in the normal ear might have been suspected *a priori.* For it is common knowledge that continuous auditory stimulation in the case of the musician, for instance, results in making the sense of hearing keener. Also that sound quality is dependent upon continuous hearing of a sound. Neither of these properties of normal hearing could be conceived possible were the auditory nerve ends subject to fatigue and corresponding alterations in refractory period found in other sense organs. And the unique properties of the auditory end organ find a counterpart in the unique anatomic structure of the ear and of the organ of Corti. Nowhere

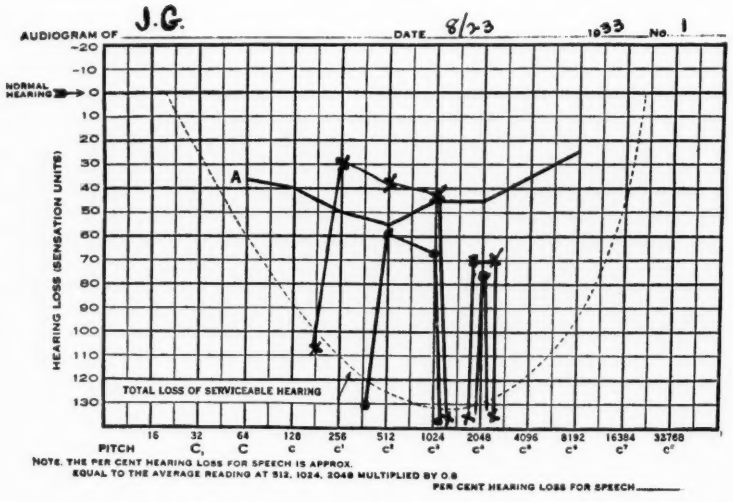


Fig. 4. x—x Right ear. .—. Left ear. Bilateral and symmetrical island of deafness between 1000 and 2000 cycles. Island of hearing about 2000 varies symmetrically from time to time; ranging from 2 to 4 notes in the right ear and from 1 to 2 notes in the left ear.

in the body is there to be found an end organ so richly supplied and bathed in nutrient medium as is the cochlea.

On the *a priori* grounds above stated, Dr. Fletcher's[9] volley theory appears to be untenable. More serious objection to the volley theory can be offered on clinical grounds. If it should be true that the auditory nerve, like other nerves of the body, were only capable of transmitting frequencies of the maximum order of 1,000 per second, and higher frequencies transmitted only by virtue of a group of fibers transmitting alternate cycles, disorder of this extremely complex sensory system should be fairly common. Such disorder would result in a perception of frequencies other than the stimulating frequencies, or in a jumbling of frequencies. This is, however, one of the rarest conditions met with in clinical otology. Dr. A. A. Gray[10] has reported one case discovered in an active otologic experience extending over many decades. As regards the apparent experimental support of the volley theory in the case of the cat, reference is again made to the finding of Dr. Hallowell Davis,[11] quoted in the first part of this paper. He reported that, with deep anesthesia, nerve action currents paralleling the stimulating sound in the cat were found only for frequencies as high as 700 cycles; whereas, under light anesthesia they were found to extend to frequencies as high as

1,000 cycles. Also, with improvements in technic and apparatus, Dr. Davis has extended his estimate of true action currents in the auditory tracts from a range of 1,000 cycles to approximately 3,000 cycles. It is possible that further refinements of technic and improvements of apparatus may remove this prop from under the volley theory.

Recent studies impel me to advance the following theory of the action of the cochlear mechanism. The high resistance offered by the capillary scalæ to the passage of sound waves make it appear highly probable that sounds of even the highest intensities could not pass through their length without great distortion and damping; and the transmission of low threshold intensities appears to be an utter impossibility. The finding of cochlear spread currents which so closely parallel in their properties the stimulating frequencies lends color to the view that the cochlea is a mechanism for the conversion of the mechanical energy of sound to electrical audio-frequency, the exact nature of which mechanism is not known, but which appears to localize at the portal of entry of sound into the cochlea and appears to be higher in its efficiency than any such mechanism now understood and known. Thus the direct stimulus of the nerve ending is predicated to be electrical audio-frequency current. The structures of the organ of Corti may well be a series of electrical filters, and not mechanical filters of sound energy as is now assumed.

On the basis of this theory the absence of auditory electrical currents in the albinotic animal could as readily be interpreted as due to ultramicroscopic, physicochemical changes in the membranes of the cochlea as in terms of degeneration of the organ of Corti.* The rising threshold of progressive deafness would signify either a diminished efficiency of the converter mechanism or a higher impedance of the receptors and neural conductors induced by altered vascularity or other pathologic changes. The accepted explanation of the more or less slowly rising threshold in the course of the advance of progressive deafness, a process which extends over a period of many years—i. e., degeneration of the auditory nerve—is hardly compatible with our knowledge of the relative rapidity of nerve degeneration.

The perception of electrical frequencies as sound has been demonstrated by several observers, including Jellinek.[12] In Jellinek's experiment hearing was obtained without the direct stimulus of sound

*Since the above was written, Dr. J. Guttman has reported, in the Journal of Physiology, October, 1934, the finding of cochlear spread currents after section of the cochlear nerve, bearing out the idea that they do not arise in the organ of Corti, but from the cochlea as a whole.

by passing through the head of the subject high radio frequencies modulated by sound, or audio frequencies. Though two alternative explanations of this phenomenon are possible—first, that the current by condenser action induces vibration of the middle ear parts, and second, that the current directly stimulates the auditory neural mechanism—the latter appears to be the more probable explanation. Further studies now under way seem to bear out the above stated hypothesis.

The "non-linearity" of hearing would not constitute any serious objection to such a theory. There is at hand sufficient data to indicate that "non-linearity" may be imparted to the hearing, in some measure, by the accommodative mechanism of the middle ear; and it is more than probable that sensory stimuli from that mechanism play a rôle in the perception of intensity and loudness. Scott N. Reger[13] has demonstrated that this mechanism, especially the tympanic membrane, is the site of end organs involved in the feeling of threshold. Von Bekesy has shown that an increase or decrease of pressure of air in the external auditory canal of 10 cm. of water results in an almost equal decrease in the loudness of intensities, especially in the frequency ranges of 100 to 1,000 cycles. His conclusion, taking into consideration other data, is that for the lower frequencies the tympanic membrane vibrates as a whole, whereas for the higher frequencies it vibrates in segments. Troger's study of the impedance of the ear drum, above quoted, shows that this impedance differs for various frequencies. He attributes to the higher impedance offered to low frequencies a protective function. This has been borne out by some of my earlier experiments on ears with perforated tympanic membranes. Stimulation of these ears with high intensities of low frequencies caused profound vertigo and nystagmus. The absence of paradoxical drop in threshold in cases with defective ear drums further bears out the rôle played by the auditory accommodative mechanism in destroying the linearity of auditory response. It also indicates that the development of auditory acuity is one of the functions of the accommodative mechanism of the middle ear.

SUMMARY.

1. Evidence is advanced of the indefatigability of the auditory end organ in the human ear, based on studies of the variation in threshold of intensity. These findings indicate that prolonged stimulation with submaximal intensities, designed to fatigue, result, on the contrary, in an increased acuity of hearing manifested by a lower threshold. It is shown that this paradoxical phenomenon is not obtained in ears in which the auditory accommodative mechanism of

the middle ear is defective; and that to this mechanism is there to be attributed, in part, the increased acuity of hearing acquired by the trained ear.

2. Fatigue manifested by a rise of threshold of intensity is described as the earliest sign of progressive deafness.

3. A theory of the mechanism of hearing is advanced which offers more adequate explanation of the observed phenomena than do the mechanical theories now most widely accepted. This theory predicates the conversion of the mechanical energy of sound into electrical energy by the cochlea; and the stimulation of the end organs of the organ of Corti by the electrical audio-frequencies thus produced. The "cochlear spread currents" are thus interpreted as the direct stimulus giving rise to the action currents of the auditory nerve.

An exception to the manifestation of auditory fatigue in cases diagnosed progressive deafness was found in a group of cases which presented varied neurologic findings. The most consistent of these neurologic findings was the head-neck past-pointing sign. These cases showed no rise in threshold following prolonged stimulation, indicating that the deafness was of central nervous type. These tests offer a differential between peripheral and central nerve deafness. With their aid the author has been enabled to isolate a type of case which on encephalogram show deformation of the ventricles but no definitely localizable lesion, which respond to spinal puncture and injection of air with a marked improvement in hearing, some cases with a restoration of hearing to almost normal, and a clearing up of tinnitus, with disappearance of the head-neck past-pointing sign and other neurologic findings. This will constitute the subject of a future publication.

993 PARK AVE.

REFERENCES.

1. Laird, D. A., and Muller, C. G.: Sleep, John Day & Co., p. 97.

2. von Bekesy: Physikalische Zeitschrift, 1928-1930.

3. McDonald and Allen: Philosophical Magazine, ser. 7, v. 9, p. 827.

4. Davis, Saul, Lurie and Derbyshire: Abstract American Otological Society Transactions, Archives of Otolaryngology, v. 18, p. 706.

5. Wever and Bray: Journal of Experimental Psychology, v. 13, p. 373.

6. Josephson, E. M.: Laryngoscope, 1929.

7. Troger, J.: Physikalische Zeitschrift, v. 31, p. 26.

8. Pavlov, I. P.: Conditioned Reflexes, Oxford Press, 1927, p. 330.

9. Fletcher: Speech and Hearing, Van Nostrand, 1929.

10. Gray, A. A.: Eye, Ear, Nose and Throat Monthly, 1930.

11. Davis and Derbyshire: American Assoc. for the Adv. of Science, Section on Psychology, Boston, Dec. 28, 1933.

12. Jellinek, S., and Schreiber, T.: Wiener Klin. Wochenschrift, April 3, 1930, v. 80.

13. Reger, Scott N.: Abstracts of Proceedings, American Acoustical Society, Dec. 4-5, 1933.

XCI.

THE SIGNIFICANCE OF THE LARYNX AS AN INDEX IN THE TREATMENT OF PULMONARY TUBERCULOSIS.

CHARLES RUBENSTEIN, M. D.,

SAN FRANCISCO.

The problem of the treatment of laryngeal tuberculosis becomes more and more closely involved with the treatment of pulmonary tuberculosis in general. There is, at present, no question that both diseases are interrelated. The modern laryngologist attacks the question of the therapy of the diseased larynx from only one angle—i. e., from that of pulmonary tuberculosis.

This article is written for the purpose of calling the attention of the laryngologist to the vast significance which the diseased larynx may have in following the course of clinical tuberculosis. The larynx may act as an index from which it is possible to view the response of the organism to the various methods of treatment introduced.

Pottenger,[5] in his valuable treatise on tuberculosis, views the problem of laryngeal tuberculosis from the modern viewpoint. We find it particularly gratifying that the conclusions reached from our findings coincide with his ideas. In discussing tuberculous laryngitis he especially emphasizes the significance of the larynx as an index in the treatment of pulmonary tuberculosis.

This is exemplified by the following excerpts:

"Tuberculous laryngitis offers an ideal opportunity for observing the development and progress of tuberculous infections.

"The importance of courage and persistence in the treatment of tuberculosis cannot be better impressed on one than by observing the larynx."

The larynx reacts sensitively to all the dynamic changes taking place in the lungs. In some cases of bilateral tuberculosis of both the lungs and larynx, when it is impossible to determine the more active side of the lungs during the examination, the pathologic findings in the larynx are beneficial in making a proper diagnosis. Hence this may prove helpful in guiding the laryngologist in the solution of one of the most vital and complicated problems in clinical tuberculosis.

It is generally believed that in most unilateral cases the affected side of the larynx corresponds to the affected side of the lungs.

Various theories have been advanced to account for this phenomenon. Pottenger attributes it to the trophic changes which are reflexly produced through the laryngeal nerves when the lungs are chronically inflamed. Schaefer[7] is inclined to the opinion that the pressure upon the recurrent nerve as well as upon part of the enlarged bronchial and tracheal glands is responsible for this condition.

Frankel explains it by the atrophy of the muscular fibers with waxy or fatty degeneration and to the inflammatory process in the nerves themselves.

Lockhard[8] assumes that the corresponding side of the larynx is first invaded because of a constitutional predisposition and weakness of the entire affected side.

The larynx may serve as an additional index of progress following the introduction of various methods of treatment.

The various stages of inflammatory reaction which take place in the larynx in the course of the different kinds of therapy introduced produce changes in the color of the larynx. The inflammatory reactions minutely parallel the immunopathologic state in the larynx. Where radical changes occur in the condition of the lungs, major changes in the throat, such as appearance or disappearance of some pronounced pathology, may occur with astounding rapidity.

It is, of course, a trying task to keep pace with the slightest change in the patient's throat as it takes place. It calls for special training, long experience, great skill, the power of concentration and a good memory.

In order to facilitate the interpretation, we have modified the hemoglobin colorimetric scale. In this way color changes in the larynx may be given a definite numerical value. We consider this scale convenient and easy in its application.

The chart may be attached to the patient's chair, or to the wall at a level with the patient's face, and the physician can view both the throat and the scale at the same time. The intensity of the color of the particular part of the patient's throat being examined can then easily be compared with the chart, and the number on the chart, to which it most closely corresponds, may be noted down. This notation forms a concrete basis for comparison with future findings.

This method may prove especially serviceable for observing progress under vaccine therapy.

CONCLUSIONS.

1. Improvement in the larynx is more likely with a concurrent general improvement in the lungs.

2. The slightest increase in the protective forces of the body, i. e., the increase of the patient's immunologic index, immediately reflects itself in the picture the larynx presents as a result of the treatment instituted for the diseased lungs. When the processes taking place in the organism, as the result of treatment, elude all other physical findings (the X-ray or any other method of observation), then the larynx will act as a faithful index.

3. To facilitate the work of following the changes as they occur in the diseased larynx, we advise the hemoglobin colorimetric scale described above.

I want to express my gratitude to Dr. F. Pottenger and his medical staff for the opportunities furnished me to study their work at close hand.

I also wish to thank the medical staff of the Los Angeles Sanitorium for the interest they have always manifested in the progress of my work.

391 SUTTER STREET.

REFERENCES.

1. Bloch, George B.: The Problems of Phthisiolaryngology, Its Clinic and Therapy. The Publications of the Tuberculous State Institution of the City of Yalta, Crimea, 1930.

2. Dobromylsky, Filipp: Voprosy Tuberculeza. Moskva. Gossudarstennoye Medizinskoye Izdatvelstvo. VII, 4:28.

3. Lockhard, Lorenzo B.: Tuberculosis of the Nose and Throat. 1909. C. V. Mosby Medical Book and Pub. Co.

4. Meriemson, H.: Voprosy Tuberculeza. Gossudarstennoye Medizinskoye Izdatvelstvo. IV, 1930. Moskva.

5. Pottenger, Francis Marion: Clinical Tuberculosis. II, 1917. C. V. Mosby Co., St. Louis.

6. Rubenstein, Charles: The Treatment of Laryngeal Tuberculosis. Calif. and West. Med., 23, Nov., 1930.

7. Schaefer, H.: Die Elektrischen und Absorptionsmessungen und Biologischen Substanzen der Ultrakurzwellen Strahlentherapie. 44:588-587, 1932. Frankf. a. M.

8. Schwartz, I., and Rubenstein, Charles: Vrachebnoe Delo. Charkoff. Gossudarstennoye Medizinskoye Izdatvelstvo. XI, 28.

XCII.

OBSTACLES ENCOUNTERED IN ELECTROCOAGULATION OF TONSILS.

WILLIAM J. YONKER, M. D.,

OAK PARK, ILL.

We were in the attitude of mind to expect favorable results from electrocoagulation of tonsils. The reason for this was the spreading of propaganda by radio, clinics provided by manufacturers, and the reports in certain medical writings and in the press. So we undertook to give this method an unbiased investigation.

Several members of our staff investigated this method in the clinic and in their private practice. We have used various types of machines. We have watched operators demonstrate the method in clinics given for that purpose and observed some utilizing the method in their private practice. We have had physicians limiting themselves to physical therapy assist us in certain cases and have had representatives of manufacturers of physiotherapy apparatus advise us as to the proper use of the machines.

Our results have been unsatisfactory and we are still in doubt whether there are any indications for this method of removal of tonsils. In the Surgical Clinic of North America (Oct., 1932, Vol. 12, No. 5, pages 1157-1171) Drs. G. E. Shambaugh, C. L. Dougherty and I have reported on our experiences with electrocoagulation in the practice of otolaryngology as it was investigated in the Department of Otolaryngology, Rush Medical College. Since that report we have continued to follow up those cases and find no reason for altering our minds in respect to the conclusions there expressed.

Among the difficulties connected with this method may be mentioned the pain occurring during and after the treatments, the length of time that a patient must be under treatment causing an unnecessary delay in the removal of pathologic tonsils, the complication of secondary bleeding, and especially the uncertainty as to whether all pathologic tissue has been removed. In the series of cases that we are reporting now we are not satisfied that all tonsillar tissue has been removed from a single case. Moreover, we have had opportunity in the clinic of examining patients who have had their electro-

coagulation done elsewhere and in not a single instance have we found that the tonsils have been eradicated.

Contrary to what is often affirmed, pain is rather prominently present both before and after treatment by electrocoagulation. By topical applications of anesthetic solutions, such as cocain, nupercain and butyn, one cannot render the throat insensitive to this method. We have attended a clinic by an electrocoagulation enthusiast who first injected a nupercain solution about the tonsils, then proceeded to dissect them free anteriorly, and finally plunged a needle through various parts of the entire tonsil in an effort to eradicate it in one treatment, but one would hesitate to justify such radical procedures. Indeed, since waterlogged tissues tend to diffuse electrical currents, we would hesitate to inject anesthetic fluids about tonsils previous to electrocoagulation because of the fear of damaging the tissues about the tonsils, particularly the large vessels. It is true that the patient does not note very much pain during the early treatments when one confines himself to the tonsillar tissue and remains at a considerable distance from the pillars. The nearer that one approaches the capsule, however, the more pronounced is the pain. Besides the pain experienced, the patient is disturbed by the noise of the machine, the increased salivation and especially the gagging.

The pain following the treatment varies with the sensitiveness of the patient, the thoroughness of the treatments, and the nearness that the so-called tonsillar capsule is approached. In one case the patient noted pain for four days following the treatments; in one, pain was sometimes felt as long as seven or eight days following applications; and in another case the discomfort complained of by the patient and the inflammatory reactions noted by the physician following only one application were fully equivalent to the discomfort experienced following surgical enucleation. If one bears in mind that patients must be treated from twelve to twenty-four times or more before one can hope to obliterate the tonsils completely, it will readily be realized that the total amount of suffering experienced by the patient will be rather significant.

Another obstacle encountered is the length of time required to complete the series of treatments. If a patient has both tonsils treated at one time and comes in regularly once a week and requires only twelve treatments for complete obliteration, three months will be required to complete the series of treatments. If one side is treated at one sitting the patient will have to come regularly for a period varying from two months to one-half year or more. The method

requires a great amount of valuable time of both the patient and the physician.

This method unduly delays the removal of diseased tonsils. Where one can trace certain ailments, such as endocarditis, nephritis, acute inflammatory rheumatism or iritis to diseased tonsils, it would be far better to remove this menace to health in one sitting than by a long series of treatments. In one case the patient developed an attack of acute follicular tonsillitis following the first treatment, something that could have been prevented if the tonsils had been enucleated by the usual surgical procedure.

It has been argued that the initial treatments by electrocoagulation tend to sterilize the tonsils, a propostiion that we are not willing to grant. Infection in tonsillar tissue may be deep seated. Crypts in them may be scarred over as a result of these treatments, or they may be so deep that they are not reached by these first treatments. In case one there were attacks of tonsillitis occurring even after twenty-four treatments had been given.

The fear of bleeding induces many patients and some physicians to look favorably upon electrocoagulation, but it must not be forgotten that this may be a complication of electrocoagulation. If one argues that this complication is a result of overzealous treatment, one might reply that bleeding following surgical removal of the tonsils is also usually a result of some indiscretion in technic. The fact is that bleeding may occur as a result of electrocoagulation. We submit that it is easier to control secondary bleeding in tissue that is healthy than in tissue that is partly coagulated and possibly friable.

The most serious obstacle which we encountered in electrocoagulation of tonsils is the extreme difficulty of completely eradicating all diseased tissue by this method. One case had ten treatments to each tonsil, another had nine treatments to each tonsil, and another had twenty-four treatments, and still all the tonsillar tissue was not removed. The reason for this difficulty is that as one approaches the so-called capsule it is hard to distinguish between scar tissue, fibrous lymphoid tissue and lymphoid tissue embedded or covered by scar tissue. Sometimes the base of the fossa appears mouse-eaten, and one wonders whether the elevations are lymphoid masses embedded in scar tissue or whether the depressions are cavities in the underlying muscle tissue.

We may continue to experiment with electrocoagulation, but, because of these various obstacles, our experience with this method of removing tonsils has thus far been disappointing.

6018 W. Roosevelt Road.

XCIII.

THE IMPORTANCE OF RADICAL ETHMOIDECTOMY AND SPHENOIDECTOMY IN THE RELIEF OF GENERAL AND OCULAR DISEASES.*

OSCAR WILKINSON, M. D.,

WASHINGTON.

The importance of meticulous nasal surgery for the relief of chronic infections has not been appreciated until very recently. In fact, during the past decade there has been so much criticism of all so-called radical surgery that the public, and even many physicians, look with much misgiving upon any procedure which smacks of radicalism.

I am entirely in accord with Ferris Smith,[1] when he suggests that we should altogether abandon the word "radical" in connection with our operations and call the procedure "complete." It expresses more accurately what we wish to say. One certainly would not call the complete removal of the faucial tonsils radical. Why then, call the complete exenteration of the ethmoid labyrinth radical, when that procedure becomes necessary? The late Ross Hall Skillern[2] very severely criticised radical ethmoid surgery, in spite of the fact that he was its master. He said:[2] "It will be seen that to resort to extensive surgery at this time with the hope of radical cure is the acme of fatuity, but something must be done, and very quickly at that, if we hope to retain the confidence of our patient." The question naturally arises, did he do complete ethmoid surgery? He usually advocated intranasal surgery and of this he says:[2] "I am also quite aware that even with this structure (uncinate process) out of the way *the entire ethmoid labyrinth cannot be removed endonasally,* but as a matter of fact that is almost never indicated, as all the cells are not always affected, at least to the same extent as to require complete exenteration." Who can tell which cell or cells are not diseased in a patient with chronic ethmosphenoiditis of years' standing? Who would want to take the chance of leaving one or two cells with the hope that they are probably not too badly diseased?

---

*Read before the Section on Oto-Laryngology of the Pan-American Medical Congress, March 24, 1934.

I feel certain that the lack of complete removal of all diseased tissue in sinus operations has caused the widespread criticism of all sinus work.

The type of case which I wish to discuss is the one showing some ocular or general infection which has not been relieved either by local remedies or intranasal surgery.

There are many chronically infected sinuses which are causing such general diseases as asthma, chronic indigestion, arthritis, low blood pressure, various cardiac diseases, renal diseases, high blood pressure, arteriosclerosis and anemia, which can be materially relieved by the eradication of the nasal focus of infection.

Of the serious ocular diseases which have been caused by these sinus infections may be included ulcer of the cornea, chronic iritis, uveitis, opacities of the vitreous, episcleritis, choroiditis, retinitis, ocular hemorrhage, degeneration of the ocular blood vessels, optic neuritis and neuroretinitis. Nothing short of the complete surgical cleansing of these sinuses will give any assurance of relief. This may be called radical, but is in fact conservative. It certainly conserves or preserves the health of the eye, and in many instances the life of the individual.

It is generally recognized that chronic infection of the ethmoid and sphenoid sinuses is associated with a similar infection of the antrum and very frequently of the frontal. If the clinical and X-ray examinations show these to be involved, they can all be dealt with at the same time. It is my practice, however, in debilitated and old patients, to do a Caldwell-Luc operation on the antrum, to wait thirty days, and then to operate upon the ethmoid, frontal and sphenoid.

I have nothing new to offer in operative technic. I use the method originally devised by Jansen,[3] in 1893. Knapp[4] described this procedure in 1899, and Guisez[5] in 1906, but we are indebted to the ingenuity of the lamented Lynch,[6] of New Orleans, for perfecting and promulgating the procedure. The finishing touches were given by Sewall,[7] who used local anesthesia and devised suitable retractors and instruments for tying the blood vessels. These aided materially in keeping the field dry, permitting a most exact inspection of all parts, and enabling the surgeon to remove all diseased tissues with the greatest safety and thoroughness. One could not discuss this subject without expressing admiration and appreciation of the most excellent technic and ingenious instruments developed by Dr. Ferris Smith for this operation. The retractors and tyer devised by Luongo of Philadelphia are very useful and worthy of

mention.[8] One would infer that this operative procedure is not generally known or practiced, for many of the newest textbooks on the subject do not mention it.

The preparation of the patient is very important. Of course no major operation should be undertaken in the presence of an acute respiratory infection. The general condition of the patient should be determined and the patient put in the best possible surgical condition. It is advisable to give 3 grs. of sodium amytal the previous night and repeat it one hour before the operation. Thirty minutes before operating a hypodermic injection of ¼ gr. morphin sulphate with 1/150th of scopolamin is given. I find this combination allays the patient's fear and aids very materially in preventing pain and keeping the patient composed.

It is essential for the operator to procure the cooperation of the patient. The best way to do this is not to hurt him, which is accomplished by the use of a 1 per cent procain injection to which is added 10 drops to the ounce of epinephrin (1/1000). Injections are made to block the supra-orbital and infra-orbital nerves, the anterior nasal nerve and the anterior and posterior palatine nerves. A local infiltration of the area of the incision is made. Proper regard must be had for the angular vein in this injection. Deep injection is made into the upper third of the orbit to block the ethmoid nerve. In this injection the needle should hug the ethmoid and frontal bones and always be kept above the ethmofrontal suture to avoid injury to the ethmoid vessel. It is advisable not to use too much injecting fluid, as it will cause excessive edema.

Intranasal application of 10 per cent cocain with adrenalin and phenol solutions are now made to the ethmoid and sphenoid regions, using small cotton-tipped applicators. The region of Meckel's ganglion is treated with this solution.

The patient is placed in a semi-sitting position, his face painted with Lugol's solution, and then wiped off with alcohol. The eyelids are sutured together with one or two silk sutures. The interior of the nose is thoroughly cleansed and swabbed with an alcoholic solution of acetone. The vestibule in particular is swabbed with Lugol's solution and the excess removed with alcohol. The patient is now draped with a dark sheet, which has an opening just sufficient to expose the nose and mouth.

The operative technic has been so well described by Lynch,[6] Sewall[7] and Ferris Smith[1] that any description by me is unnecessary, but I would like to emphasize a few points. A well taken X-ray plate will be of great aid in determining the extent of the cells. The

size of the ethmoid and frontals will determine the length of the skin incision. A 3 cm. incision is sufficient to begin with. All vessels should be tied at once with a fine catgut in order to keep the field dry. The periosteum should not be elevated over the mesial side of the incision, as Lynch has shown that the incidence of osteomyelitis is lessened when the periosteum is not elevated.

In eviscerating the lining membranes of the various cavities the use of gauze covered curettes, or better still, gauze balls, as recommended by Smith, is indicated. This operation requires both gentleness and thoroughness. No diseased membrane, overhang or crevice must be left. After the cleansing I use Lugol's solution, removing the excess with tincture of benzoin. Every care should be taken in closing the wound to procure cosmetic results.

SUMMARY.

It is becoming more generally recognized that a certain number of chronic, serious, general and ocular diseases are due to chronic sinus infection.

The older so-called radical intranasal and extranasal operations have been in disrepute chiefly because they were too destructive, on the one hand, and not sufficiently complete on the other. With this operative method one can eliminate sinus infection in practically every case.

This operation can be done with a minimum of sacrifice of the tissues and with a minimum of danger and discomfort to the patient.

1408 L STREET, N. W.

REFERENCES.

1. Smith, F.: Management of Chronic Sinus Disease. Arch. Otolaryng., 19: 157 (Feb.), 1934.

2. Skillern, R. H.: The Ethmoid Problem. ANNALS OF OTOLOGY, RHINOLOGY AND LARYNGOLOGY, 37:173 (Mar.), 1928.

3. Jansen, C.: Arch. Laryng. u. Rhin., 1:135, 1893.

4. Knapp, A. H.: Arch. Ophth., 28:50, 1899.

5. Guisez: Ophth. Congres de Paris, 1906.

6. Lynch, R. C.: Laryngoscope, 21:162 (Jan.), 1921.

7. Sewall, E. C.: External Operation on the Ethmosphenoid-frontal Group of Sinuses Under Local Anesthesia. Arch. Otolaryng., 4:377 (Nov.), 1926.

8. Luongo, R. A.: An Eye Retractor and a Suture Carrier Knot-tier for the External Pansinus Operation. Arch. Otolaryng., 18:4 (Oct.), 1933.

Clinical Notes and New Instruments.

XCIV.

A WOOD TONGUE DEPRESSOR IN THE TRACHEA THIRTEEN YEARS: CHRONIC LARYNGEAL STENOSIS.*

GABRIEL TUCKER, M. D.,

PHILADELPHIA.

The unusual features of the following case make it seem worthy of presentation. They are as follows:

1. One-half of a wood tongue depressor was aspirated into the tracheobronchial tree of a young woman, seventeen years of age, following tonsillectomy.

2. The patient and physicians suspected that the foreign body was in the trachea, but the patient refused to have an examination and removal of the foreign body until thirteen years later when it became impacted in the subglottic larynx. The laryngeal obstruction became so great that intervention with removal of the foreign body was necessary to relieve the dyspnea.

3. Following the removal, dyspnea was temporarily relieved with the subsidence of the swelling in the larynx. One month later the stenosis had increased to such an extent that tracheotomy was required for relief of the laryngeal obstruction that had developed.

REPORT OF A CASE.

CASE 1.—A well developed and nourished female, aged thirty years, a stenographer by occupation, was referred to me for relief of urgent dyspnea. The patient's sister, who is a trained nurse, stated that two weeks before admission, while the patient was engaged in a game of baseball, she was making a home run and just as she reached the home plate she fell with a choking attack, becoming urgently dyspneic. After considerable difficulty her dyspnea was partially relieved and she was taken to Dr. David Robb of Ithaca, New York, who made a diagnosis of stenosis of the larynx as the result of the foreign body being coughed from the trachea up into the larynx from below. (Fig. 1.) The dyspnea persisted, becoming more marked until the patient was unable to sleep, when she decided to come in and have the foreign body removed.

*Read before the annual meeting of the American Laryngological Association, Cleveland, Ohio, June 7, 1934.

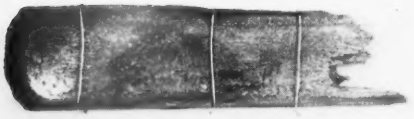


Fig. 1. Photograph of the portion of tongue depressor removed by direct laryngoscopy from the subglottic larynx after thirteen years' sojourn in the tracheobronchial tree of the patient.

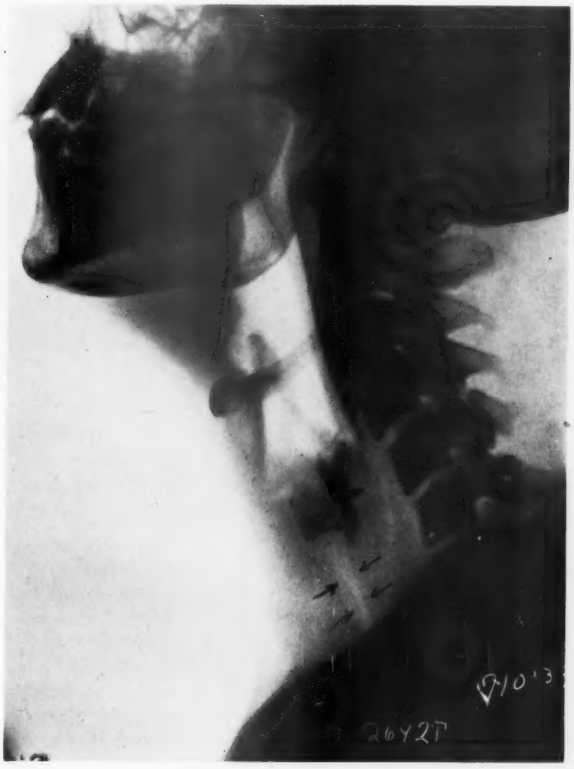


Fig. 2. Lateral roentgenogram of the neck showing a narrowing of the lumen of the subglottic larynx with inflammatory reaction in the region of the impacted foreign body. (Before removal.)

Fig. 3. Lateral roentgenogram of the neck four days after removal of the foreign body. The swelling had subsided to such a degree that dyspnea had largely disappeared. The patient was allowed to return home. One month later cicatricial contraction occurred to such an extent that tracheotomy was required for relief of the dyspnea. Patient was treated by direct laryngoscopic bouginage and at the present time there is an adequate airway through the larynx.

The patient states that "following the tonsillectomy, thirteen years ago, after she had been returned to bed, she was supposed to have choked and swallowed her tongue. Efforts were made to pull the tongue forward with a wood tongue depressor and the tongue depressor broke and half of it disappeared." At the time the physician in attendance told the patient that it probably went into her trachea, but she refused to have anything done about it. She states that during the thirteen years she had been unable to lie on her left side because in doing so urgent dyspnea occurred. When she would turn on her right side or back the dyspnea would be immediately relieved. She states that she "felt something moving in her trachea on cough and it seemed to block her left lung when she would lie on her left side." On admission the patient was urgently dyspneic and on mirror examination the broken end of the portion of tongue depressor could be seen in the lumen of the subglottic

larynx. Direct laryngoscopy was done and the foreign body removed without difficulty. (Fig. 2.) The dyspnea was immediately relieved. A very small bronchoscope, 3.5 mm. by 35 cm., was passed and the trachea and tracheobronchial tree were inspected. There was noted inflammatory reaction in the trachea and right main bronchus, indicating that the foreign body had been freely movable previous to its impaction in the subglottic larynx. The laryngeal reaction subsided and at the end of three days the lumen of the subglottic larynx seemed to be at least three-fourths that of normal. (Fig. 3.) The patient was allowed to go home under the observation of Dr. Robb. She remained at rest and for a period of three weeks from the time of the removal her condition seem to improve. At the end of this period, however, signs of increasing dyspnea developed and mirror examination showed obstructing tissue in the subglottic area. The patient was sent back by Dr. Robb, and on direct laryngoscopy fungating tissue and marked thickening in the subglottic area was found. A low tracheotomy was done and the dyspnea was entirely relieved. Tissue was subsequently removed from the subglottic larynx and was reported by Dr. E. A. Case as follows: "Inflamed granulation tissue." Direct laryngoscopic dilatation was carried out after the tracheotomy wound had healed and further tissue was removed which was again reported as chronic inflammatory tissue.

Direct laryngoscopic dilatation has been continued and at the present time the patient has almost an adequate airway through the larynx. The mucous membrane is perfectly smooth and there is no evidence of ulceration or cicatricial tissue.

COMMENT.

The foreign body, one-half of wood tongue depressor, remained in the tracheobronchial tree for a period of thirteen years, being freely movable and produced only a slight inflammatory reaction in the mucous membrane. It was coughed into the subglottic larynx and became impacted and produced sufficient reaction to cause marked stenosis of the larynx. Following the removal of the foreign body the patient developed a chronic subglottic laryngeal stenosis which could be ascribed only to the trauma of the foreign body. The chronic stenosis has responded to rest, due to the low tracheotomy and repeated direct laryngoscopic bouginage.

326 SOUTH 19TH ST.

XCV.

A VALVULAR TRACHEOTOMY TUBE.*

GABRIEL TUCKER, M. D.,

PHILADELPHIA.

The occurrence of a greater degree of obstruction on inspiration than on expiration in certain conditions of the larynx, particularly bilateral posticus paralysis, makes a tracheotomy tube possessing a valve that obstructs the expiratory flow of air desirable. In 1930, I devised such a tube. It has been in use for the past four years but has not as yet been officially published. Because of the simplicity and adaptability of this tube it is now presented.

A patient who requires a tracheotomy tube over a long period ordinarily has very little thickening in the front of the neck so that a tube of medium curve and length is desirable. For this reason a curve shorter than the Jackson tube is used. The valve mechanism is made entirely in the inner tube so that when the inner tube with the valve attachment is in place the outer tube acts as the valve seat. This permits the valve being made of such size that it is impossible for it to pass through the inner tube, should it become loosened, into the tracheobronchial tree. The mechanism is quite simple and has proved to be very effective. (Fig. 1.) The tube is also provided with the usual inner tube that the patients may use when they do not desire to have the valvular tube in place. The valve can be adjusted so that only sufficient air is forced through the larynx to produce a voice. If the stenosis of the larynx is too great for the full amount of air to pass, part of the return flow may come out through the tube.

326 SOUTH 19TH ST.

---

*Presented before the annual meeting of the American Laryngological Association, Cleveland, June 7, 1934.

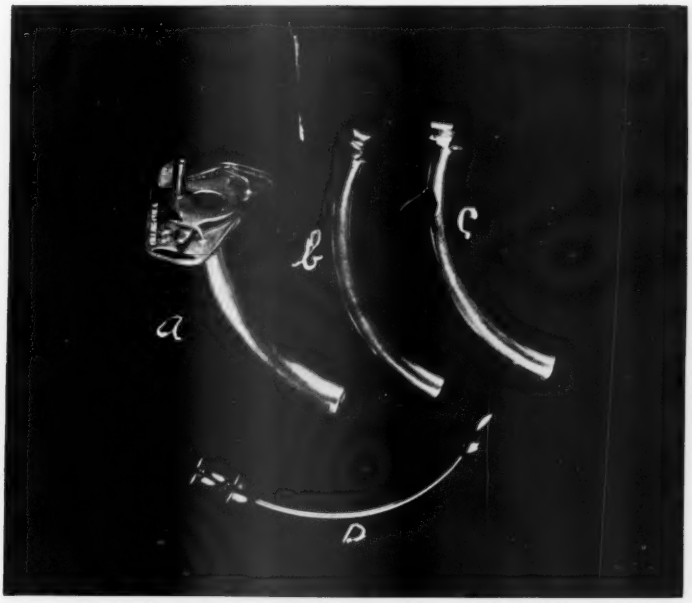


Fig. 1. A Valvular Tracheotomy Tube. *a*, Outer cannula of special length and curve. *b*, The standard inner cannula. *c*, Inner cannula provided with valve. *d*, Obdurator for use for insertion of tube. The valve *c* is so constructed that it will not pass through the lumen of the tube should it become loosened.

XCVI.

A KNIFE FOR THE TREATMENT OF WEB STENOSIS OF THE LARNX.*

GABRIEL TUCKER, M. D.,

PHILADELPHIA.

The usual treatment of web stenosis is incision or dilatation or a combination of these procedures and possibly thyrotomy and excision in certain cases.

Recently I have treated a case of congenital web where these methods were not feasible. Gentle bouginage caused so much reaction in the larynx that a method of incision of the web with spreading of the cut surfaces was used. A knife was devised which is a combination of dilator and cutting instrument with a protected point.

A sharp cutting blade is placed about 1.1 cm. from the distal end of a rod, the rod being 30 cm. in length from its tip to the attachment of the handle. In using this knife through the direct laryngoscope the rounded tip distal to the cutting blade prevents trauma to the subglottic larynx and trachea. The blade of the knife is triangular from the base towards its edge. As the rod proximal to the cutting edge is drawn outward from below the web the margin of the web is stretched at the point where it is to be incised. As the cutting edge is withdrawn through the web the rounded end of the tip of the knife follows and separates the cut margins. The passage of this knife through the incision opens it with very little trauma if there has been a tendency for the cut margins to reunite. It is to be used every second or third day until the cut surfaces epithelize. The cutting edge keeps the incision open and the rod-like tip and shaft dilates the incision without exerting too great pressure.

326 SOUTH 19TH ST.

*Presented before the annual meeting of the American Laryngological Association, Cleveland, June 7, 1934.

A Knife for the Treatment of Web Stenosis of the Larynx. The instrument is 30 cm. in length and is for use through a direct laryngoscope. *a*, Shows the cutting edge on the upper surface. *b*, Shows the projecting tip that prevents injury to the trachea when the blade is pushed against the web. The instrument acts both to incise the web and can be used to dilate gently the incision while the cut margins are epithelizing.

XCVII.

A TRACHEOTOMIC ETHER INSUFFLATION TUBE.*

GABRIEL TUCKER, M. D.,

PHILADELPHIA.

A number of years ago I devised a tracheotomic insufflation tube. I am presenting a modification of this tube. The tube is made in three sizes, three, five and seven. (Fig. 1.) It may be used in the open end of the trachea at laryngectomy and for ether insufflation through a tracheotomy wound for operative procedures in the larynx or pharynx. The modification consists of enlarging the caliber of the inflow tube and placing a cap over the proximal end of the tube to which a rubber tube is attached in order to carry away the ether vapor that is blown outward by the patient on expiration.

326 SOUTH 19TH ST.

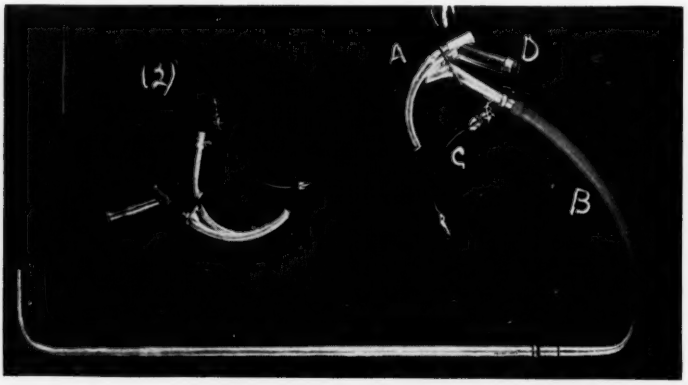


Fig. 1. A Tracheotomic Ether Insufflation Tube. *1*. Large size insufflation tube for intratracheal use after laryngectomy. *a*, Insufflation tube. *b*, Inflow vapor tube which is attached to the insufflation apparatus. *c*, Obturator for insertion of tube. *d*, Cap to which a rubber tube is attached to carry the exhaled ether vapor away from the field of operation. *2*. The same set of smaller size for insufflation through a tracheotomy fistula.

The apparatus is constructed in two sizes. One for insufflation into the trachea after laryngectomy, the other for insufflation through a tracheotomy fistula. A third smaller size is also made for children.

*Presented before the annual meeting of the American Laryngological Association, Cleveland, June 7, 1934.

The Scientific Papers of the American Bronchoscopic Society.

XCVIII.

PRIMARY COLLOID ADENOCARCINOMA OF THE LOWER THIRD OF THE TRACHEA.*

W. Likely Simpson, M. D.,

Robert M. Moore, M. D.,

Memphis.

Compared with their frequency in the larynx, primary malignant tumors of the trachea are surprisingly rare. The majority of cases are not recognized until so far advanced that treatment is of little if any avail. Although the region of the bifurcation is considered to be the most common site, the literature through December, 1933, reveals only twenty-seven cases of primary carcinoma of the lower third of the trachea, including the authors' case.

REVIEW OF THE LITERATURE.

In an extensive review of the literature dealing with primary tracheal tumors, D'Aunoy and Zoeller,[1] in 1931, were able to find but nineteen cases of carcinoma of the lower third of the trachea. One of these cases, that reported by Adam,[2] in 1915, was reported again by him[3] in 1926, thereby leaving only eighteen cases collected by D'Aunoy and Zoeller. Gilfoy,[4] in 1932, collected five additional cases, one each reported by Breslich,[5] Neilson,[6] Chevallier,[7] and two by Figi;[8] and reported one case of his own. Also, Vinson and Leddy,[9] and Teuber,[10] in 1933, each reported a case.

REPORT OF A CASE.

Case 1.—E. P., a white female, aged 34 years, was admitted to the medical service of the Memphis General Hospital, July 1, 1933, and came to autopsy on the following day with the clinical diagnosis of bronchial asthma.

*From the Departments of Otolaryngology and Pathology, University of Tennessee, Memphis, Tenn.

Read before the seventeenth annual meeting of the American Bronchoscopic Society, Cleveland, June 11, 1934.

Fig. 1.

Fig. 2.

Fig. 1. Intratracheal portion of tumor, showing sessile origin from right posterolateral wall just above bifurcation. Trachea and bronchi opened posteriorly; aortic arch, left; left main bronchus and part of right, below. Photograph.

Fig. 2. Cross section of tumor showing involvment of entire thickness of tracheal wall with encroachment upon lumen. Aortic arch, left; left main bronchus, below. Photograph.

The illness had begun one week prior to admission with a head cold followed by shortness of breath, a sense of constriction in the chest, and the inability to breathe comfortably while lying on the left side. The past history revealed two similar attacks, one lasting a week and the other of five months' duration; occurring during the past two years.

Postmortem Examination: The lumen of the trachea, just above its bifurcation, was almost obliterated by a moderately firm, smooth, lobulated mass attached in a sessile manner to the right posterolateral wall, protruding externally to lie adjacent to but separated from the right tracheobronchial lymph nodes by a delicate connective tissue capsule. The intratracheal portion of the mass measured 2x1½x1 cm. in its various diameters and was covered by an intact mucous membrane (Fig. 1). The extra-tracheal portion measured 3x2½x1½ cm. On section (Fig. 2), the mass was moderately firm, grayish-white and translucent, but contained numerous small spaces filled with a gelatinous substance.

The respiratory passage above the mass was not unusual. Below, the passages were filled with a tenacious mucopurulent exudate which covered an edematous and congested mucous membrane. The bronchi and bronchioles were moderately dilated and thick-walled. Each lung contained areas of consolidation surrounded by emphysematous tissue. The tracheo-bronchial lymph nodes were not unusual.

The remaining pathologic findings at autopsy were irrelevant, and no secondary metastasis from the tracheal mass could be found.

Microscopic Examination: An intact stratified, columnar, ciliated epithelium covered the intratracheal portion of the mass, which was composed of large and small groups of hyperchromatic epithelial cells having a marked glandular arrange-

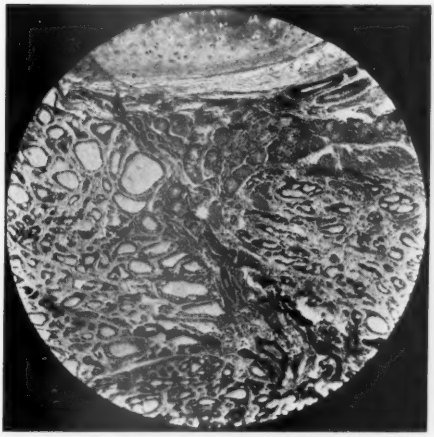


Fig. 3. Intratracheal portion of tumor, showing origin of carcinoma from mucous glands of submucosa, center. Cartilage, above; glands distended with mucous, left. Photomicrograph x160.

ment, arising from the mucous glands of the submucosa (Fig. 3). Mitotic figures were not numerous. Many of the glands were distended into cyst-like spaces lined with cuboidal epithelium and filled with a grayish-pink, stringy material. These cells and glands had invaded the entire thickness of the tracheal wall, surrounded the cartilaginous rings and composed the extra-tracheal portion of the mass. Here the glands were even more markedly distended and widely separated by a loose, edematous stroma, and in some areas the individual cells were loose, spherical and contained droplets of clear fluid in their cytoplasm (Fig. 4.) The extra-tracheal portion was covered with a loose connective tissue capsule which was continuous with the capsule of an adjacent lymph node. The latter structure showed an accumulation of small round cells and polymorphonuclear leucocytes, but was entirely free from invasion by the malignant cells composing the tumor (Fig. 5).

Anatomical Diagnosis: Primary colloid adenocarcinoma of the lower third of the trachea causing partial obstruction of the trachea, resulting in emphysema and bronchiectasis, bilateral; chronic tracheobronchial lymphadenitis; and bronchopneumonia, bilateral.

DISCUSSION.

Of the twenty-seven cases recorded in the literature, including the authors' cases, seventeen occurred in males. In twenty-two instances the patients were between the ages of forty and sixty-four years.

The posterior wall, which is richer in glandular element than either the anterior or lateral walls, was the most common site of the tumors, although the exact location was not stated in nine cases. The lateral wall, relatively rich in glandular structure, was involved

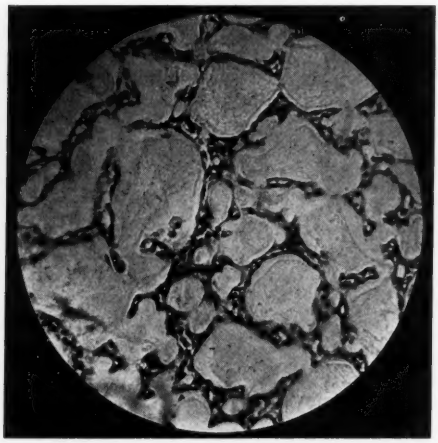


Fig. 4. Section from central portion of tumor showing marked colloid degeneration. Photomicrograph x500.

either solely or in combination with the anterior or posterior wall in numerous cases, and in two instances the tumors almost completely encircled the trachea.

The widespread metastasis occurring in seventeen of the twenty-seven cases is in direct contradistinction to laryngeal carcinoma, which rarely metastasizes, and shows a marked predilection of metastasis to a limited number of organs. Because of their frequency in the membranous portion of the trachea the tumors frequently grow posteriorly to involve the esophagus, but the mucous membrane of the latter rarely shows ulceration. The tissues involved in the present series, in the order of their frequency are, the regional lymph nodes, peribronchial tissues, liver, esophagus, lungs, cervical lymph nodes, pleura, spleen, retroperitoneal lymph nodes, kidney, pancreas and skeleton.

In eleven instances the duration of symptoms was less than one year. The onset was either abrupt or insidious, and, as a rule, exertional dyspnea resembling "asthma" was the primary symptom noted by the patient. The attacks of dyspnea were usually periodical, and were often influenced by the patient's position; lying on one side or the other would often produce marked relief, or the recumbent position would greatly aggravate the respiratory difficulty, which was either inspiratory or expiratory in nature. In the interval between attacks the patient would be free of symptoms. A tickling sensation in the throat, leading to cough and occasional hemoptysis or hoarse-

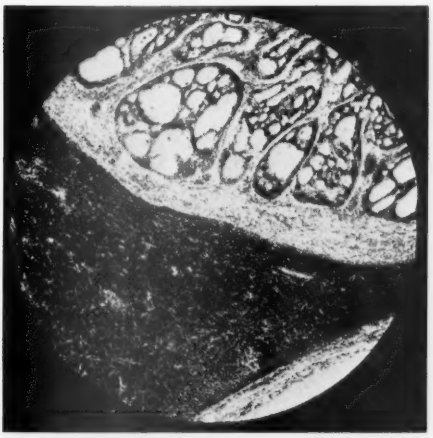


Fig. 5. Marginal section of extratracheal portion of tumor, showing adjacent lymph node, left, free of metastasis. Photomicrograph x160.

ness may be the presenting symptom, but usually the latter occurs late in the disease as a result of laryngeal nerve involvement. Cachexia was not often noted, probably because of the relative short duration of the disease, although in some instances dysphagia was a marked symptom, due to early involvement of the esophagus.

In regard to the histogenesis of these tumors, D'Aunoy and Zoeller, quoting von Langhans, Hamacher, Virchow and others, express the belief that the origin is in the mucus glands, but in the case of squamous cell tumors are inclined to the view of development from cell rests rather than to the theory of metaplasia, and call attention to the fact that the respiratory system develops from the anterior foregut. From the fact that the trachea is lined with stratified columnar epithelium, in connection with which there are numerous mucus glands, Figi anticipated finding glandular carcinoma the most common type of malignant tumor arising in the trachea. This is not borne out from our review of carcinomas arising in the lower portion of the trachea. Histologically, as reported, eight of the tumors in the present series were of the squamous cell variety and one was a basal cell tumor, whereas, only eleven cylindric cell or adenocarcinomas were mentioned. It is true that on account of the great difference in terminology and lack of microscopic data on the cases recorded in the literature, these cases are extremely difficult to classify histologically. It is believed, however, that the tumor reported by us is the only tracheal carcinoma showing colloid degeneration.

SUMMARY AND CONCLUSION.

A review of the literature through December, 1933, reveals but twenty-seven cases of primary carcinoma of the lower third of the trachea. The majority of these cases were not recognized until so far advanced that treatment was of little avail. Such a case, with extensive colloid degeneration, discovered at autopsy, is reported.

The combination—dyspnea, apparent good health, absence of intrathoracic signs—suggests tracheal tumor and demands bronchoscopy.

We are indebted for advice to Dr. Harry C. Schmeisser, Director, and for photographs and photomicrographs to Mr. J. G. J. Perkins, Jr., Photographer, University of Tennessee, Pathological Institute.

512 PHYSICIANS' AND SURGEONS' BLDG.

BIBLIOGRAPHY.

1. D'Aunoy, R., and Zoeller, A.: Primary Tumors of the Trachea: Report of Case and Review of Literature. Arch. Path., 11:589-600 (April), 1931.

2. Adam, John: Case of Tracheal Tumor. J. Laryng. and Otol., 30:64-66 (February), 1915.

3. Adam, John: Four Cases of Tracheal Tumor. J. Laryng. and Otol., 41:174-177 (March), 1926.

4. Gilfoy, F. E.: Primary Malignant Tumors of the Lower Third of the Trachea: Report of Case with Successful Treatment by Electrofulguration and Deep X-Rays. Arch. Otolaryng., 16:182-187 (August), 1932.

5. Breslich, P. J.: Squamous Cell Carcinoma of the Trachea. J. Cancer Research, 14:144-151 (March), 1930.

6. Neilson, D. F. A.: Case of Primary Carcinoma of the Trachea. J. Laryng. and Otol., 45:855-858 (December), 1930.

7. Chevallier, R.: Primary Carcinoma of the Trachea Beginning with Dysphagia. Lyon Med., 146:309-315 (September 14), 1930.

8. Figi, F. A.: Primary Carcinoma of the Trachea. Arch. Otolaryng., 12:446-456 (October), 1930.

9. Vinson, P. P., and Leddy, E. T.: Carcinoma of the Trachea. Proc. Staff Meet., Mayo Clinic, 8:641-643 (October 25), 1933.

10. Teuber, K. H.: Primary Cancer of the Trachea. Ztschr. f. Hals-, Nasen- u. Ohrenh., 33:444-458, 1933.

XCIX.

BRONCHOSCOPY IN TUBERCULOSIS.*

MERVIN C. MYERSON, M. D.,

NEW YORK.

A review of the literature and a perusal of textbooks on bronchoscopy and tuberculosis reveals that very little has been written and not much is known concerning bronchoscopy in tuberculosis. It is natural that the subject of bronchoscopy as it relates to the tuberculous patient should have received little if any attention. An impression has long existed that only a few cases of tuberculosis of the lung justify bronchoscopy. There has been an attitude of caution in the matter of bronchoscopy for these patients, which is pardonable because it has been based upon fear of possible consequences rather than upon actual knowledge of what to expect. It is felt that with the usual care in the selection of cases, there is no harm in performing bronchoscopy upon tuberculous individuals. Although all cases of tuberculosis do not require this procedure, it would seem that a fair number present such clinical symptoms, physical signs or x-ray findings as not only to justify bronchoscopy but actually to require it.

There are occasional reports of bronchoscopies which have been performed for special indications where an unsuspected tuberculous lesion has been found, but few bronchoscopies have been performed on patients who were known to have or were suspected of having the disease. It is to be expected that a disease such as tuberculosis with so great a variety of manifestations would at times present problems to the clinician which would lead him to seek the assistance of the bronchoscopist. Surgery and artificial pneumothorax have proven to be added factors in the creation of a need for bronchoscopy, for at times these procedures so alter conditions within the chest as to confound the clinician.

What cases of tuberculosis then require bronchoscopy? In the first place there are the same general indications in tuberculous individuals as there are in those with nontuberculous pulmonary disease. Then there are cases which present indications that might be consid-

---

*From the Department of Otolaryngology, Sea View Hospital, N. Y.

Read before the seventeenth annual meeting of the American Bronchoscopic Society, Cleveland, Ohio, June 11, 1934.

ered directly related to the disease. The cases which have been bronchoscoped could be classified according to indication as follows:

1. Those presenting obstructive phenomena.
   Tuberculous obstructive lesions.
   - A. Tuberculoma.
   - B. Fibrotic or cicatricial.
     1. Endobronchial.
     2. Peribronchial.
   - C. Caseous bronchitis and thrombus formation.
   - D. Caseous gland.

   Nontuberculous obstructive lesions.
   - A. Mucoid material following pneumothorax.
   - B. Mucopus following thoracoplasty.
   - C. Carcinoma of the bronchus.

   Non-obstructive lesions.
2. Hemoptysis of unexplained origin.
   - A. Tuberculous ulceration.
   - B. Carcinoma of the bronchus.
   - C. Benign ulceration of the bronchus.
3. Asthmatic or asthmatoid breathing.
   - A. Compression of lower trachea or main bronchus by glands.
   - B. Evidence of unsuspected mediastinal tumor.
4. Localization of disease for the surgeon.
   Indications peculiar to the tuberculous patient:
5. Persistence of positive sputum when surgery or pneumothorax should have
   - A. Caused the sputum to become negative.
   - B. Decreased the amount of sputum.
6. Effort to secure a positive bacteriologic specimen from the branch bronchus which brings the exudate into the main bronchus.

This paper deals with bronchoscopies performed upon sixty adult patients who were known to have pulmonary tuberculosis or were strongly suspected of having the disease. These cases are taken from the records of the past year. Most of them were performed at the Sea View Hospital, a large tuberculosis hospital. Almost all the remaining bronchoscopies were done at the Metropolitan Hospital (tuberculosis service), while a few were seen in private practice. This paper does not include a fairly large number of bronchoscopies which were performed upon patients who were not known to have or were not suspected of having tuberculosis. The cases which were

bronchoscoped were considered as being tubercular for one of the following reasons:

1. Positive sputum with positive physical and x-ray findings.
2. Positive sputum with negative x-ray and physical findings.
3. Negative sputum with positive x-ray and physical findings.
4. Previous history of positive sputum.

Are there any constant findings in the bronchial tree of the tuberculous? Perhaps the picture that appears with the greatest frequency is that encountered in cases of long standing disease where there is evidence of fibrotic changes within or outside of the bronchi. The endobronchial fibrotic lesion is that of a true cicatricial stenosis which may be only partly occlusive but usually completely obstructs the bronchus. The peribronchial fibrotic lesion evidences itself in a greatly increased thickening of the dividing spur and a consequent obliteration of or a diminution in the size of the bronchial branch orifices. When a pale mucosa was encountered it had no special significance, for it partook of the same pallor which characterized all the other tissues because of the secondary anemia from which the patient was suffering. In none of the cases of this series was there evidence of erosion brought about by peribronchial or peritracheal lymph masses. Broadening of the carina was not a frequent finding, and compression of the tracheobronchial angle by an enlarged lymphatic gland was noted three times.

The bronchoscopic findings are exemplified in the following case reports:

REPORT OF CASES.

CASE 1.—Tuberculoma of the bronchus. P. C., male, age 54, had pain in left side eleven months; shortness of breath, eleven months; cough; expectoration, eleven months. X-ray showed slight retraction of the left chest with localized diminution of aeration of the left base. There were productive changes at the roots of both lungs.

The sputum was positive for tubercle bacilli.

Bronchoscopy, by Dr. Herman Rubin, showed the left main bronchus to be narrowed in its lower portion. An irregular mass was found occluding the left lower lobe bronchus. A small mass of granulations was seen in the right lower lobe bronchus.

The laboratory report was: Tissue from left bronchus, tuberculoma. Tissue from right bronchus, chronic inflammatory.

CASE 2.—Caseous thrombus of bronchus. L. C., female, age 19, had productive cough two months; pain in left chest for two weeks, and fever for two weeks.

Physical signs were decreased fremitus, dullness, distant breath sounds in left upper lobe region.

X-ray showed infiltration of the left upper field. Shadow suggested sacculated empyema or atelectasis.

Bronchoscopy showed a thick, irregular, friable, whitish, exudate completely filling the upper lobe bronchus of left side. Impression: Tuberculous caseous thrombus.

The laboratory report was necrotic material with many tubercle bacilli.

CASE 3.—Right main bronchial obstruction following thoracoplasty. A man of 50 was operated upon by Dr. Paul Coryllos. An extensive thoracoplasty for tuberculous disease was performed on the left side. Thirty-six hours after operation he experienced severe dyspnea and an elevation of temperature. The right side of the chest was almost completely atelectatic.

Emergency bronchoscopy revealed that the right main bronchus just below the upper lobe branch was occluded by a large amount of thick viscid mucopus. The patient was definitely relieved by the bronchoscopy.

CASE 4.—Tuberculous ulceration of lower trachea and right main bronchus. G. G., female, age 34, complained of cough, expectoration, and hoarseness for five months.

X-ray report showed caseous pneumonic lesion of the right upper lobe region. Pneumothorax had produced an excellent collapse.

The patient was referred by Dr. Ornstein because despite the excellent collapse there was no improvement in cough, hoarseness or sensation of choking, and because the sputum continued positive. Dr. Ornstein suspected ulceration or local bronchial lesion.

Bronchoscopy showed superficial irregular white elevations in the midst of a yellowish depressed surface in the right lower tracheal wall laterally and extending downward into the right main bronchus where a definite ulceration was seen. Specimens were removed from both areas.

Impression: Tuberculous ulceration.

The laboratory reported the specimen from the bronchus too small to interpret. The specimen from the trachea was chronic granulation tissue.

NOTE.—Occasionally such tissue as the above can be shown to contain tubercle bacilli if specially stained. This should be done whenever tissue is submitted to the laboratory for a possible diagnosis of tuberculosis.

CASE 5.—Ulceration of bronchus; tuberculous nature not proved. S. M., female, age 46, had hemoptysis for three years, cough for five years, and expectoration for five years.

Diagnosed as tubercular before admission.

X-ray showed diffuse productive changes throughout the right lung and left lower lobe region with marked thickening of the pleura in the right upper lung field. There was an annular shadow at the right apex which might be a cavity.

Bronchoscopy was performed by Dr. J. G. Gilbert. Exudate was seen coming from the right upper lobe bronchus. A small ulcer about five millimeters in diameter, which bled readily, was seen on the floor of the left main bronchus, just below the carina.

Biopsy report: Tissue from ulcer edge—chronic inflammatory tissue.

CASE 6.—Benign ulceration of the bronchus. I. S., female, age 28, complained of dry cough, hemoptysis, slight fever and chest pains for nine months.

X-ray showed exudative productive changes in the left apex and infraclavicular region. Exudative productive changes in the right upper lung with coalescent exudative lesion in axillary portion of second right interspace.

Bronchoscopy by Dr. J. G. Strickler showed a superficial healing ulceration on the floor of the left main bronchus, about four millimeters in diameter. This bled readily when touched.

CASE 7.—Benign ulceration of left upper lobe bronchial spur. Patient referred by Dr. Charles Kaufman. J. S., age 30, male, had cough, expectoration and frequent blood streakings for two years, and an artificial pneumothorax for the past two years.

Sputum was positive until six months ago.

X-ray showed a well established pneumothorax in the left chest. The cavity in the left upper chest was apparently obliterated.

Bronchoscopic examination showed thick mucoid material coming from the left upper lobe bronchus. On the upper aspect of the upper lobe bronchial spur there was a small superficial erosion which bled readily when touched.

CASE 8.—Fibrotic stenosis—endobronchial. A. W., male, age 25, complained of cough for two years, expectoration for two years, loss of weight for two years, dyspnea for one year. A thoracoplasty had been performed two years ago.

X-ray showed a partial collapse of the right chest. There was a large uncollapsed cavity in the right infraclavicular region.

Sputum was positive.

Bronchoscopy by Dr. H. Rubin showed introitus of the right upper lobe bronchus completely stenosed by scar tissue. Dilation was attempted but was not satisfactory.

CASE 9.—Fibrotic stenosis—peribronchial. O. R., male, age 52, had the following symptoms: Productive cough for three years, hemoptysis three years ago, loss of weight for two years, weakness for two years and vomiting for two months.

X-ray showed a caseous pneumonic lesion of the right upper lobe and an exudative lesion at the root of the right lung. There was slight retraction of the right upper chest, and a slight deviation of the trachea to right. Neoplasm of the right upper lobe bronchus was suspected.

Bronchoscopy by Dr. H. Danish showed the orifice of the right upper lobe bronchus reduced to almost a pin point in size. No endobronchial lesion was seen.

CASE 10.—Fibrotic stenosis—peribronchial. M. H., female, age 25, complained of cough for five years, expectoration for four years and pain in left chest one year.

X-ray showed asymmetry of the chest; the left side was smaller than the right. Deviation of the trachea and heart to the left. The left pleura was thickened. There was no aeration of the left lower lobe. The left upper lobe was incompletely aerated.

Bronchoscopy by Dr. Wm. J. Hochbaum showed orifices of the bronchi on the left side much smaller than those on the right. The dividing spurs were much thicker than normal; this was very marked in the left lower lobe bronchus, the branch bronchial orifices being almost completely obliterated.

CASE 11.—Fibrotic stenosis—peribronchial. A. H., male, age 47, complained of productive cough, dyspnea, loss of weight and pains in right chest for four years.

X-ray showed a caseous pneumonic lesion involving the right lung. The right lower lobe showed no aeration. The heart and trachea were shifted to right. There was a caseous pneumonic lesion of the left upper lobe.

Bronchoscopy by Dr. H. Rubin showed right recurrent laryngeal nerve paralysis. The carina was thickened and pulled over to the right. The lumen of the right main bronchus was narrowed. The orifices of the right lower lobe bronchus were occluded by marked thickening of spurs.

CASE 12.—Atelectasis due to peripheral fibrosis; all bronchi patent. H. R., male, age 36, complained of cough and expectoration for many years.

Sputum was positive.

X-ray showed the left lung atelectatic. Trachea and heart were deviated to the left.

Bronchoscopic examination showed all branches visible through bronchoscope were patent.

CASE 13.—Fibrotic stenosis—extensive peribronchial fibrosis. J. M., male, age 43, complained of dyspnea and productive cough for many years. There was pronounced dullness to percussion on the right side.

X-ray showed atelectasis of the right lung.

Bronchoscopy showed the bifurcation of the trachea markedly distorted. The orifices of the branches leading from the left main bronchus were markedly dilated, about twice their normal size. Introitus of right main bronchus was a horizontal slit. The surrounding tissue resisted the passage of the bronchoscope through the slit. The branch bronchi of the right side were closed and could not be entered. There was no evidence of expansion or contraction on the right side during respiration. There were no bronchial lesions.

Impression was that of a long standing peribronchial fibrosis.

CASE 14.—Obstruction of main bronchus by thick mucoid material. Atelectasis, obstructive. Patient referred by Dr. Foster Murray. F. McC., male, age 31. The history showed that the patient's father and one sister had died of tuberculosis. The patient had complained of cough, expectoration and languor for several years.

X-ray showed the trachea and heart shadows markedly displaced to right. There was a smooth homogeneous shadow overlying the entire right pulmonic field. Impression was that of extensive atelectasis of the right side.

Bronchoscopy showed the right main bronchus to be occluded by thick mucoid material which was removed by suction.

NOTE.—This patient was referred for bronchoscopy because the right lung failed to expand after withdrawal of the artificial pneumothorax.

CASE 15.—Carcinoma of the bronchus. N. S., female, age 43, had pain in the left chest for four months and cough and expectoration for sixteen months.

X-ray showed a productive tuberculosis of the right upper lung area, with massive left sided effusion.

Sputum was negative.

Bronchoscopy showed an irregular growth on the posterior wall of the left main bronchus.

Report from laboratory: Adenocarcinoma.

CASE 16.—Carcinoma of the bronchus. E. J., male, age 61, complained of weakness, cough, expectoration and loss of weight for past six months.

The sputum was always positive.

X-ray showed diffuse emphysema with a fibrocaseous tuberculosis of the left upper lobe with cavity at the clavicle region. Localized prominence of the left hilus.

At bronchoscopy a mass was encountered in the supero-anterior wall of the left main bronchus just before the branching of the left upper lobe.

The laboratory reported the specimen a squamous cell carcinoma.

CASE 17.—Alveolar carcinoma with chronic productive tuberculosis. J. M., male, age 42, had pain in the right chest, cough and expectoration and loss of weight for two and one-half years.

The sputum was negative.

Bronchoscopy showed considerable thick mucopurulent exudate obstructing the right main bronchus. There was an irregular infiltrating growth, nonobstructive, seen on the floor of the right main bronchus just above the lower lobe branching.

The report on specimen was catarrhal inflammation.

Autopsy findings were terminal (alveolar) carcinoma of the lung and chronic nodular tuberculosis with cavities in the right lung.

CASE 18.—External compression of left main bronchus with granulation like mass penetrating bronchial lumen. M. J., male, age 29, complained of cough, expectoration, occasional blood streaked sputum and languor for seven years.

Sputum was positive

The patient had a collapse of the left lung with a large ball valve cavity occupying one-third of the left lung. Physical findings, altered breath sounds, and moist rales are out of proportion to x-ray impression.

Bronchoscopy showed an anteroposterior flattening of the left main bronchus which ends in a horiozntal crease in the region of the left lower lobe bronchus. A granulomatous mass was seen in this sulcus. The bronchus was markedly dilated just before this mass was reached, the dilatation extending in the lateral diameter for about one and one-half inches. Specimen was removed.

Impression was that of external compression of the left main bronchus by a neoplasm which was penetrating into its lower portion.

Laboratory report was chronic inflammatory tissue.

CASE 19.—Tracheobronchial compression by enlarged lymph gland. Patient referred by Dr. Edgar Mayer. V. M., female, age 35, complained of asthmatic like breathing for one year.

X-ray showed an indefinite shadow in the left hilus.

Sputum was positive.

Bronchoscopy showed the left lower tracheal wall and the upper wall of the left main bronchus, at its beginning markedly indented so as to reduce the lumen to about one half of normal.

CASE 20.—Carcinoma of the esophagus. E. A., male, age 55, had cough and expectoration for one month, weakness, loss of weight and dysphagia for six months.

X-ray showed a large cavity in the right infraclavicular region and another at the root of the left lung. There were diffuse exudative and productive changes irregularly distributed throughout the upper lobes.

Sputum was highly positive.

Bronchoscopy showed exudate coming from the right upper and left lower lobe bronchi.

Esophagoscopy showed a cauliflower like growth partly occluding the esophagus twenty-eight centimeters from the upper tooth mark.

Specimen was reported squamous cell carcinoma.

Many cases showing x-ray shadows and physical signs which indicated the possibility of obstructive lesions were found to be negative at bronchoscopy. In addition we encountered a fair number of cases which were negative at bronchoscopy and which were suspected of having ulcerative lesions. These were the cases which had been operated upon or had had the benefit of extended pneumothorax therapy but continued to show a positive sputum. The surgeons or medical men wished to know whether or not an ulcerative lesion existed or whether a gland had penetrated into the bronchus.

We have encountered many patients with clinical evidence of laryngeal tuberculosis, with relatively few chest signs and with a negative sputum. It should be noted that in not a single instance was there an exacerbation of the disease or a spread to a new area of the lung following bronchoscopy. It was thought before we had done any of this work that bronchoscopy performed upon a case of suspected tuberculosis in which the sputum was negative would very likely convert the negative sputum into a positive one. This did not prove to be the case.

Hemoptysis was not the sequel in any of the cases which had experienced this symptom prior to bronchoscopy. It is our rule to wait at least ten days or two weeks after a hemoptysis before we accept a case for bronchoscopy.

Our experience would indicate that with ordinary care in the selection of cases of tuberculosis of the lung for bronchoscopy there should be no untoward effects.

SUMMARY AND CONCLUSIONS.

A series of sixty patients known to have pulmonary tuberculosis or strongly suspected of having this disease were subjected to bronchoscopy because of a definite indication in the minds of the referring clinicians, all experts in the field of chest diseases. The conditions which were encountered are related and examples cited. This study strongly indicates that bronchoscopy should have a definite place in the study of the tuberculous patient. No untoward results were seen as a result of bronchoscopy in pulmonary tuberculosis.

The writer takes this occasion to thank Dr. George G. Ornstein, director of the tuberculosis services at the Sea View and Metropolitan Hospitals, and his associates, Drs. Kaufman, Ulmar and Cherkoff, and Dr. Coryllos of the department of surgery, for their co-operation and assistance in the study of the hospital cases.

136 EAST 64TH STREET.

C.

A PROPOSED OPERATION FOR THE RELIEF OF CONGENITAL ATRESIA OF THE ESOPHAGUS.*

SAMUEL IGLAUER, M. D.,

CINCINNATI.

In the most common form of congenital atresia the esophagus is made up of two segments, a blind pouch above and a lower segment connected with the trachea through a fistulous tract near the bifurcation. As a result of these anomalies the newborn infant regurgitates all of its food, while air enters and distends the stomach by way of the trachea and lower esophageal segment. The infant dies from starvation or from a bronchopneumonia induced by a spill-over from the blind pouch into the larynx and trachea, or from the entrance of vomited gastric juice which enters the trachea from below.

The diagnosis of this anomaly can usually be made from the history and from x-ray examination after the introduction of an opaque catheter into the blind pouch. The film shows the catheter in the pouch and a markedly distended stomach. The barium meal should not be given in a suspected case, since it may enter the tracheobronchial tree and fill the lungs. I present such a film, in which a pediatrician gave barium to a moribund infant with congenital atresia. As I have pointed out in another paper,[1] in a suspected case of tracheoesophageal fistula the administration of lipiodol is preferable as an opaque medium. Reid[2] suggests the injection of lipiodol into the trachea.

In a postmortem study of a case of congenital atresia presented to this society, Freiberg and I[3] have shown that it was possible to pass a ureteral catheter through a cystoscope from the stomach through the lower segment into the trachea, which was then filled with lipiodol. We were also able to pass the catheter from below in several patients, gastrostomized for other conditions.

In a recent case of congenital atresia Tucker and Pendergrass[4] were able to show the communication between the trachea and esopha-

*Read before the seventeenth annual meeting of the American Bronchoscopic Society, Cleveland, June 11, 1934.

gus by passing a ureteral catheter through a bronchoscope into the trachea through the fistula and into the stomach.

From time to time various operative procedures have been tried for the relief of congenital atresia, but no typical case has ever been relieved by operation. Gastrostomy has frequently been performed, but has proven unsuccessful because of the regurgitation of food from the stomach into the trachea. Jejunostomy has also been tried with unsuccessful results. To prevent regurgitation Ravdin[4] tied off the esophagus below the diaphragm at the same time that he performed a gastrostomy.

Using spinal anesthesia, Sarnoff[5] exposed and freed the esophagus below the diaphragm, passed a double ligature about the esophagus, divided the same between the ligatures, and then brought the lower stump to the surface as an abdominal esophagostomy, through which the infant was fed. The patient died on the thirteenth day, succumbing to an aspiration pneumonia.

Richter[6] operated upon two cases in the following manner:

He first opened the abdomen and made a small incision in the anterior wall of the stomach, through which he introduced a pliable guide into the lower segment of the esophagus. A catheter was inserted into the trachea in preparation for ether insufflation anesthesia. The thorax (right) was then opened widely under positive intrapulmonary tension, and a ligature was passed around the lower segment at its junction with the trachea. The chest was then closed by sutures and the lung was reinflated before inserting the final suture.

This operation was immediately followed by gastrostomy. The first case died of shock shortly after operation. The second patient reacted fairly well but succumbed to pneumonia twenty hours after operation.

From his experience, Richter[6] concluded an infant could withstand the shock of this operation.

Both Richter[6] and Lilienthal[7] suggest the possibility in some cases of forming an anastomosis within the chest between the two segments of the esophagus. As emphasized by Lilienthal,[7] pneumonia will inevitably ensue unless some provision is made to prevent spilling of mouth secretions from the upper segment into the trachea. Many operative failures can be ascribed to this neglect of the upper segment.

PROPOSED OPERATION.

The operation which I wish to propose belongs in the field of thoracic surgery and, therefore, is offered with considerable diffidence.

The operator will, however, require the assistance of a bronchoscopist, and it is, therefore, proper to present the subject to this society, the members of which are usually consulted when these cases are encountered. I wish to add that I have done considerable anatomic and experimental work on the infant cadaver in the study of this subject.

The operation is planned in two stages:

I. *Cervical Esophagostomy.*—Exposure of the cervical esophagus on the right side with the establishment of an esophageal fistula to drain the buccal secretions away from the trachea. Using the classical technic, this operation can readily be performed under local anesthesia, aided by a guiding catheter inserted from above. If possible, the stoma in the esophagus should be united with the skin edges. Otherwise the esophagus can be exposed, the wound packed, and the stoma created as soon as the mediastinum has become protected by adhesions. Cervical esophagostomy should not be attended by very great shock. Unless the upper segment can be taken care of, there are practically no indications for any further intervention. Prior to and after this operation the infant should be nourished by intravenous or intraperitoneal administration of glucose solutions.

II. *Transthoracic Operation.*—This second stage operation should follow the first as soon as possible. Inhalation ether anesthesia, followed by insufflation anesthesia, should be employed during this operation. A specially prepared ureteral catheter should be inserted through the tracheal fistula into the stomach, according to the method of Tucker.[4] This catheter should serve the double purpose of conveying ether vapor to the lungs and of acting as a guide to the esophagus when the chest is opened. The tip of the catheter should be sealed with paraffin to prevent insufflation of the stomach. The catheter should be provided with a small rubber collar about eight centimeters from the tip. This collar should occlude the tracheo-esophageal fistula and prevent leakage of air from the trachea into the stomach. Just above the collar a few small openings should be provided to permit the entrance of the insufflated ether vapor into the trachea. (Fig. 1.)

Incision.—A semilunar incision is made beginning in the right posterior axillary line over the sixth or seventh intercostal space. The incision is carried mesially, and extends upward over the spinous processes to the third intercostal space. The underlying muscles are dissected for a short distance laterally, exposing the ribs in the region of their angles. The pleura is now opened through the seventh intercostal space, and insufflation anesthesia is begun with six to eight mil-

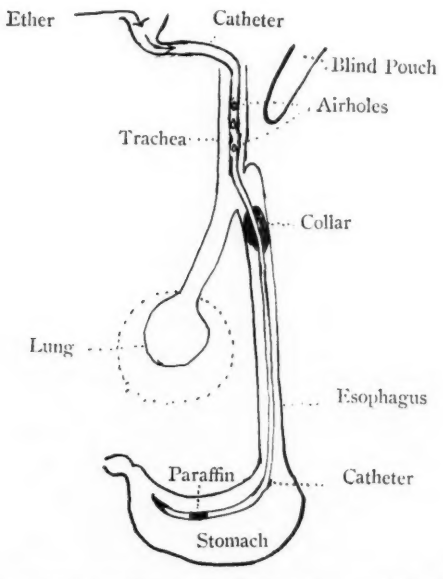


Fig. 1. Showing ureteral catheter passed through the tracheal fistula into lower segment of esophagus and into the stomach.

limeters of mercury pressure, according to Richter.[6] The ribs and underlying pleura are now divided near the rib angles, beginning with the seventh and terminating with the fourth rib.

The ribs are now carried upward and the chest held open with an automatic retractor. A flat retractor is used to push the lung upward and forward.

The azygos vein is seen ascending in the thorax under the parietal pleura, and crosses over to join the superior vena cava. The palpating finger now locates the catheter contained within the esophagus and will be found just mesial to the azygos vein. (Fig. 2.) The vein is divided between ligatures placed near its termination. The parietal pleura is divided longitudinally over the esophagus. The right vagus nerve is seen branching into the esophageal plexus.

Novocain is now injected into the periesophageal areolar tissue to block the vagi, according to the method of Heller.[8] The right vagus is carefully dissected free from the esophagus, which is loosened from its bed. A ligature is passed about the esophagus near its junction with the trachea, and the esophagus and its contained catheter is divid-

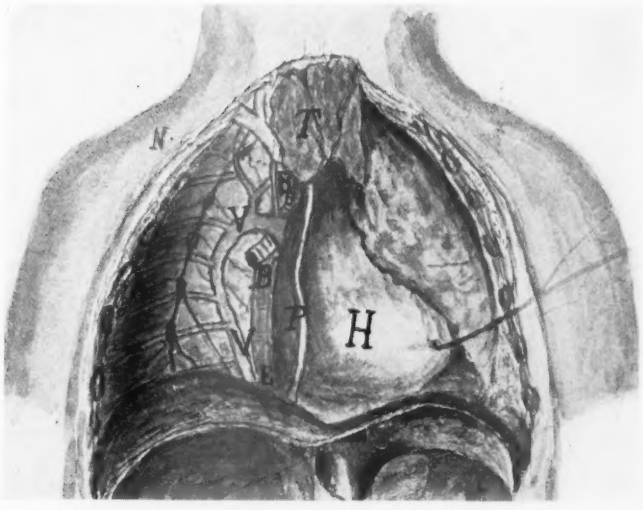


Fig. 2. New born infant. Right lung has been removed. *H* heart, *P* phrenic nerve, *T* thymus gland, *E-E'* esophagus, *N* vagus nerve, *V-V'* azygos vein. (At *V'* the azygos vein should be divided between ligatures), *B* right bronchus.

ed below the ligature. The tracheal end of the divided catheter is slightly withdrawn into the trachea and a second ligature is passed about the upper esophageal stump to prevent leakage of air.

The lower stump is then transplanted without tension, and the cut edge of the esophageal stump is sutured to the skin at a convenient point in the lower margin of the wound. (Fig. 3.) (Owing to the tenuity of the esophageal wall, suturing is difficult.) A small portion of the adjacent rib is removed to prevent pressure on the esophageal stoma. A small rubber catheter is substituted for the ureteral catheter in the transplanted lower segment, and is sutured to the skin margin to prevent its loss into the stomach. The thoracic opening is now closed by sutures, the lung being fully inflated before tying the last suture (12 to 15 millimeters mercury—Richter[6]).

Should the patient survive, feeding could be carried on through the dorsal esophagostomy. The upper and lower stomata could also be joined by a connecting rubber feeding tube, as advocated by Hedblom[9] and Torek.[10] Later in life an intervening link might be provided by a plastic anastomosis of some kind. This would permit the taking of food by mouth.

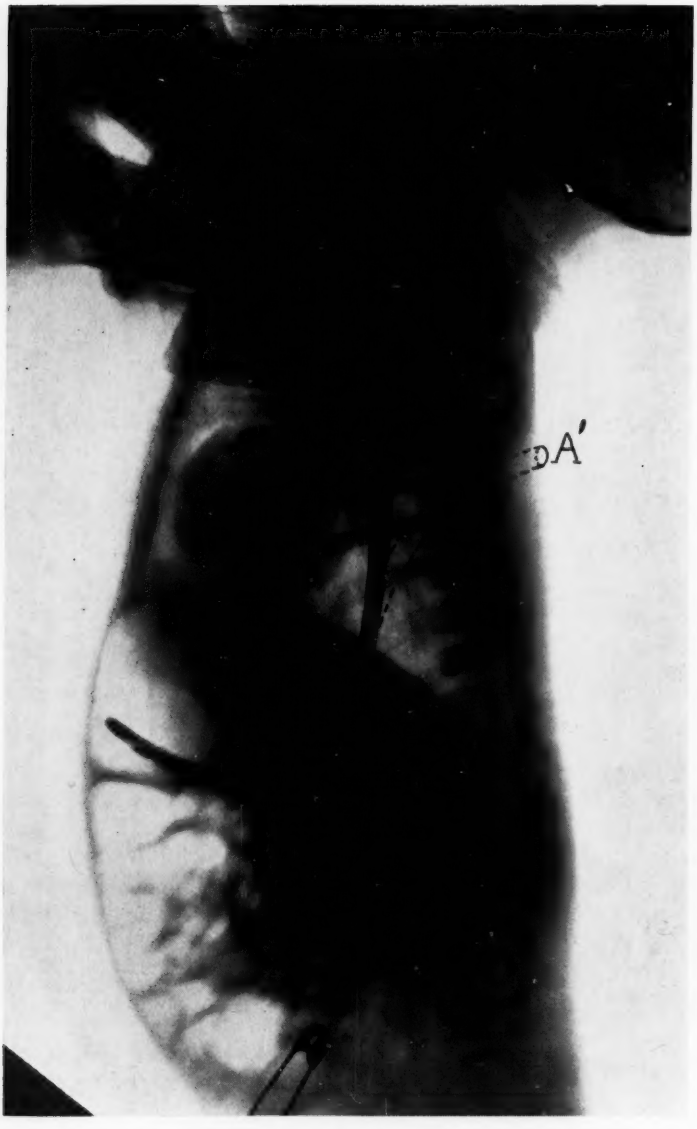


Fig. 3. Normal case with catheter in the esophagus. Roentgenographic diagram to illustrate how lower segment is transplanted in dorsal esophagostomy. *A* upper end of lower segment, *A'* same end transplanted and sutured to skin of back.

COMMENT.

This operation is proposed with the full realization that it would be a very hazardous procedure in an infant, but it would seem justifiable to make the experiment in view of the 100 per cent mortality of all previous methods which have been employed.

SUMMARY.

A two-stage operation is proposed for the relief of congenital atresia of the esophagus:

Stage 1. Cervical esophagostomy for drainage of the blind esophageal pouch.

Stage 2. A transthoracic operation for the externalization of the lower esophagus, i. e., dorsal esophagostomy for feeding purposes.

I wish to express my indebtedness to Dr. Richard Austin of the Pathological Department for anatomic material, and to Mr. Philip Goland for assistance in dissections.

707 RACE ST.

BIBLIOGRAPHY.

1. Iglauer, S.: Nonopaque Foreign Bodies in the Esophagus and Esophagotracheal Fistula: Their Demonstration by Ingested Iodized Oil. Arch. of Otolaryng., 7:229-33, March, 1928.

2. Reid, M. R.: An Aid in the Diagnosis of a Certain Type of Congenital Esophageal Stenosis. J. Ped., 1:87, 1932.

3. Iglauer, S.: Impermeable Stricture of the Esophagus Relieved by Retrograde Bouginage with the Aid of a Cystoscope Inserted Through a Gastrostomy. ANNALS OF OTOLOGY, RHINOLOGY AND LARYNGOLOGY, 40:1191 (Dec.), 1932.

4. Tucker, G., and Pendergrass, E.: Congenital Atresia of the Esophagus. J. A. M. A., 101:1726, 1933. Ravdin, I. S.: Quoted by Tucker and Pendergrass.

5. Sarnoff, J.: Atresia of the Esophagus in the Newborn: System of General Surgery in Motion Pictures.

6. Richter, H. M.: Congenital Atresia of the Esophagus: An Operation Designed for Its Cure. S. G. O., 17:397-403 (Oct.), 1913.

7. Lilienthal, H.: Thoracic Surgery. Vol. 1, p. 321, Philadelphia, 1925.

8. Heller: Quoted by Rehn, E.: Operationen am Oesophagus. Handbuch d. Hals- Nasen- u. Ohrenheilkunde, Band 9, p. 424, 1929.

9. Hedblom, C. A.: S. G. O., 35:284, Sept., 1922.

10. Torek, F.: Esophagectomy. Cyclopedia of Medicine, Vol. V, p. 433, 1932.

CI.

NOTES ON ESOPHAGUS CASES.*

HARRIS P. MOSHER, M. D.,

BOSTON.

This paper consists of the presentation and discussion of four instruments, a discussion of fibrosis of the esophagus, illustrated by histologic slides, and of two cases of stricture of the esophagus. Both strictures were caused by caustics; both had a large pouch at the upper end of the esophagus with the opening of the esophagus excentric. In one of the cases the stomach had been pulled into the chest half way to the arch of the aorta. Both cases were treated by fluoroscopic dilatation with metal tipped bougies.

THE MERCURY BOUGIE.

The mercury bougie is a rubber tube made in the form of a bougie and, as its name implies, is filled with mercury. I first learned of it from the medical side of the hospital where they were using it in treating cardiospasm.

The great advantage of the bougie in my hands is that it can be given a patient after the dilatation of the fibrosed cardia has reached a certain point. The patient soon learns to pass the bougie on himself. The blunt point prevents all danger of perforation. The weight of the bougie carries it down almost of itself. The expense of the bougie ($7.50) is a bit prohibitive, especially to hospital patients.

In dealing with fibrosis of the esophagus, I have had two great surprises. The first was the finding that the flexible metal finder, the size of which is about 30 F., will pass the strictured area in the great majority of cases of cardiospasm, in spite of the fact that the barium picture of the esophagus shows the terminal portion apparently to be closed. The second surprise was that the small mercury bougie (No. 30), which is numbered English style and is almost twice as large as the metal spiral wire finder which I use on the diagnostic bag, will also pass in many cases of cardiospasm. One set of men handling these cases, generally the internists, maintain that the pass-

*Read before the seventeenth annual meeting of the American Bronchoscopic Society, Cleveland, June 11, 1934.

age of the larger mercury bougie proves that the lesion in the esophagus is due to spasm; the other set of men, and I belong to this small and select group, hold that a twist of the terminal portion of the esophagus plus the local fibrosis is responsible for the appearance of complete closure and that the bougie untwists this.

METAL TIPPED BOUGIES.

A metal tipped bougie shows clearly under the fluoroscope, so that fluoroscopic dilatation of many strictures is possible, provided you are assisted by a competent roentgenologist, and provided further

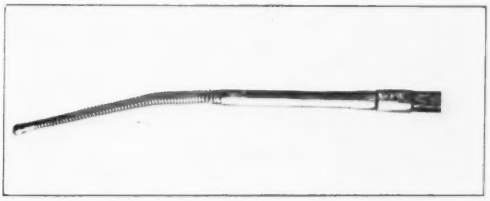


Fig. 1. The illustration shows a small flexible bougie with a cone-shaped metallic end which carries a flexible spiral wire finder. It is designed for use under fluoroscopic vision in the dilatation of small strictures of the esophagus.

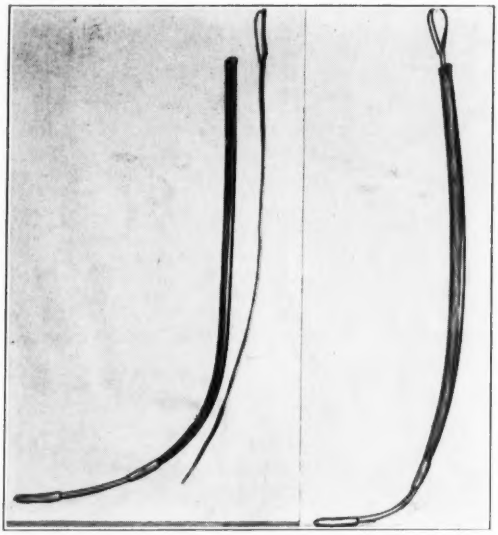
Fig. 2. The illustration shows a flexible bougie, No. 18 French, tipped with a cone-shaped metallic end. The bougie, is designed for dilatation of strictures under fluoroscopic vision.

that he is familiar with chest x-rays, and especially with the fluoroscopic examination of the esophagus. I am blest with such a man, of course, in Dr. MacMillan.

I am using two types of metal tipped bougies. The first (Fig. 1) has a special spiral wire flexible tip. This is used in the first attempts at dilating a tight stricture. The second type (Fig. 2) is solid and cone shaped. This is used after the flexible finder has located the opening of the stricture, and the stricture has been dilated to the full diameter of the shaft of the bougie carrying the flexible finder.

In beginning fluoroscopic dilatation with the flexible finder, quite a little probing often is necessary before the tip of the bougie finds the opening of the stricture because it is often off center.

It has proved to be a useful procedure in such cases to withdraw the bougie slightly, at the same time rotating it on its vertical axis, and then to carry it down again. In successful attempts the point of the bougie will suddenly drop into the opening. When the operator has once gotten the trick of finding the opening, in any given case the next attempt generally succeeds almost at the first trial.



a *b*

Fig. 3. Hollow Flexible Bougie With Wire Staff.

The first illustration shows a flexible hollow bougie fitted with a flexible spiral wire finder *(b)*. The copper wire staff used for straightening the bougie is shown in *(a)* next to the bougie.

The second illustration shows the bougie with the staff inserted. Insertion of the staff straightens out the bend of the bougie shown in the first illustration.

The flexible bougie is used in cases where the esophagus is markedly bent. The bougie with the flexible finder is inserted under the guidance of the fluoroscope and when the finder has entered the stomach the staff is inserted in the bougie. This straightens out the bend of the bougie and makes it possible to obtain a direct shot through the cardiac opening of the esophagus.

In those where the tip of the diagnostic bag would not enter the stomach, the narrowing at the cardiac end of the esophagus is dilated by the procedure just described.

Any size hollow bougie can be fitted to the flexible finder. This makes it possible to dilate under direct vision up to 40 French.

THE HOLLOW BOUGIE WITH STAFF.

In beginning the treatment of certain old cases of fibrosis of the esophagus (cardiospasm), where the terminal three or four inches of the esophagus lies flat on a motionless diaphragm and the bougie has to pass two bends (dead man's curve) and the bend at the top of the crural canal, the flexible finder will pass into the stomach but the bag will not follow. The problem is to dilate the narrowing at the top of the crural canal sufficiently to allow the bag to pass. The staff of the bag is too flexible to allow much if any force to be transferred through it to the tip of the bag. I have found it useful in this condition to pass a hollow bougie having a flexible spiral wire finder (Fig. 3). With the finder in the stomach the staff or wire is inserted in the bougie. This straightens out the curve of the terminal part of the bougie, and, of course, stiffens it. With the bougie now in line with the axis of the crural canal, it is justifiable to put considerable pressure on the wire staff and to carry the bougie through the strictured area. It is striking how an esophagus which sags markedly to the right can be straightened out by this procedure.

STAFF FOR CARRYING A THREADED BULLET.

I have many times attempted to carry a thread attached to a perforated shot through the esophagoscope and beyond a strictured area of the esophagus, only to have the shot vomited up as the patient came out of ether. I have, therefore, lately devised two shot carriers designed to be passed under the fluoroscope. The staff in the two instruments is so made that when the shot has been carried to its limit in the strictured esophagus the staff can be disengaged and leave the shot in place. (Fig. 4.)

THE LIGHT CARRYING BOUGIE.

The light carrying bougie is a bougie of the small Jackson type which has a bronchoscopic light buried in its shaft near the tip (Fig. 5). The purpose of the light is to make the bougie easier to find when it has been passed into the stomach. In such cases, of course, the stomach already has a gastric fistula, and the purpose of inserting the string is for carrying out retrograde bouginage. It is not always easy to recognize a bougie at the cardia of the stomach, nor in the stomach itself, even with a short esophagoscope carried through the gastric fistula.

The instrument I am showing is at present only a pretty toy. Its practical use is yet to be demonstrated. It was designed for an especially hard case in which a number of attempts to have the patient

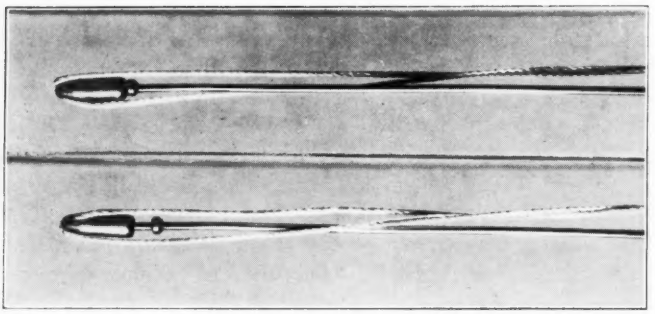


Fig. 4 shows a metallic bullet carrying a thread. The bullet is held in place on its staff by traction on the thread. Illustration *a* shows the bullet in place and in illustration *b* the bullet is partially disengaged in the staff. The thread carrying the bullet is designed to be used under fluoroscopic vision.

swallow a string had failed. The house officer (Dr. Judd), however, committed the unpardonable sin of getting a string down the day before the instrument was finished.

I have lately had two caustic strictures of the esophagus, both of which were complicated by a pouch at the upper end of the esophagus at the beginning of the stricture. In both cases the opening of the esophagus was off center. Both patients were examined under ether and the opening of the esophagus located and its size determined. The lumen of the esophagus in both cases was about two millimeters in diameter.

Any progress in dilating these cases meant either repeated etherizations or an attempt to dilate the strictures under the fluoroscope. This procedure was finally successful in both of the cases. However, at first it was hard to find the opening from the pouch into the esophagus and when it was found the dilatation was extremely slow. Naturally it was extremely anxious work, but thanks to Dr. MacMillan's management of the fluoroscope, I was able at all times to tell where the metal tip of the bougie was. The bougie used was the one shown in Fig. 1. The bougie has a tapering spiral tip about 2½ inches long. This form of tip makes it possible for the bougie to find its way around corners and, being flexible, the danger of perforating the esophagus is greatly minimized.

The first case which was dilated under fluoroscopic control was a high school boy, about sixteen, who in doing a chemical experiment at home, swallowed some alkali by mistake. He was referred by his physician to the hospital five months after the accident. At this

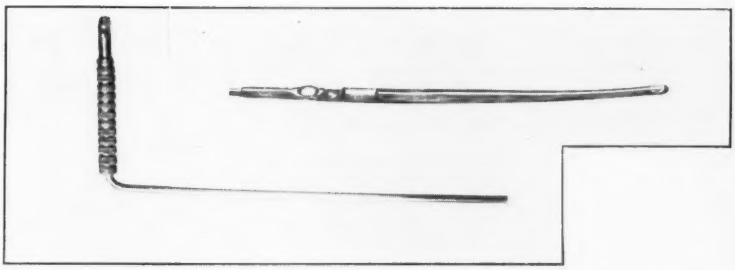


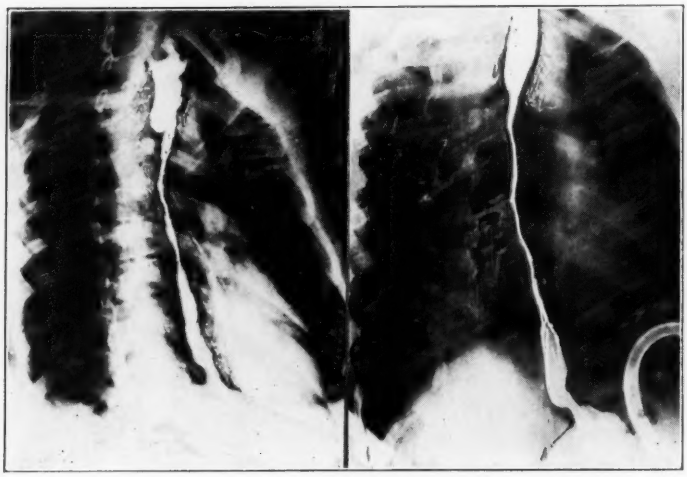
Fig. 5. The illustration shows a small Jackson bougie fitted with a bronchoscopic lamp. The bougie was designed to pass into the stomach and be picked up from there through a gastric fistula. It should be of use in locating the cardiac opening of the esophagus from the stomach and for carrying a string from the stomach through the esophagus.

time he was reduced to swallowing liquids with difficulty. The fluoroscope showed a sizable pouch in the upper part of the esophagus at the beginning of the stricture. (Fig. 6, a and b.) The stricture extended from the arch of the aorta to the middle third of the esophagus. Barium passed through the strictured area with difficulty, and it was a question whether or not gastrostomy should not be done at once for feeding purposes.

The first few attempts to find the opening of the esophagus with the spiral wire tipped bougie were failures. The attempt to have the patient swallow a string was also a failure. Finally, however, the bougie found its way into the opening of the esophagus, and after three or four trials with the bougie, gaining a little each time, it was passed through the whole of the stricture into the stomach. After the first dilatation the patient had a temperature for about a week and caused me great anxiety. However, the films of the chest showed no infection in the mediastinum and the temperature, as I said, was normal at the end of about a week. This temperature happened again in the course of the fluoroscopic dilatation of the stricture, and it was decided it was too hazardous to keep on with this manipulation and a gastrostomy was done. Following this the patient was finally successful in swallowing the string. Retrograde dilatation was then begun and has been carried on at intervals to date. The maximum size of bougie which can be passed at present is No. 28 F. The patient has gained in weight markedly and is eating everything, including meat, the meat, of course, being minced.

This case shows that the fluoroscopic dilatation of the esophagus by the use of metal tipped bougies is feasible and can be successful. It is, of course, nerve wracking work and its success depends as much

a



b *b*

Fig. 6. X-ray films (retouched) of a boy of sixteen, who swallowed caustic five months previously.

The esophagus is strictured in the middle third. At the beginning of the strictured area the esophagus is dilated into a pouch. Examination under ether showed that the opening of the esophagus was excentric and markedly to the left.

The upper figure shows a side view *(a)* of the pouch. An attempt was made to pass a thread beaded with shot through the esophagus after a bougie had been passed into the stomach by aid of the fluoroscope. The film shows the bagging of the pouch below the opening on the esophagus. Two shots have passed into the esophagus.

The first of the two lower figures *(b)* shows the strictured area and the pouch. The second figure *(c)* is from a film taken after retrograde dilation of the esophagus had been carried on some four months. The pouch has shrunken and the opening of the esophagus is not in line with the tip of the pouch.

This is the first instance in which the writer has observed this happening.

on the competency of the roentgenologist as on the skill of the operator who manipulates the bougie.

The patient in the second case in which fluoroscopic dilatation with metal tipped bougies was tried was a young woman of twenty. When she was two years old she swallowed some form of caustic and has had a strictured esophagus ever since. I saw her when she was about ten and periodical bouginage was carried out easily and successfully. Then the patient fell into the hands of a noted orthopedic practitioner of this city, who at that time had the idea, and unfortunately I think he still has it, that he can cure stricture of the esophagus by posture. I saw the patient again after some years and advised gastrostomy on account of the extreme narrowness of the stricture and on account of her poor condition. This operation was performed with the understanding that the patient was to have retrograde dilatation following the procedure. However, a thing which is new to me, but which Dr. Jones, the medical man, says he has seen three instances of, happened, namely, the gastric feeding did not nourish the patient, and it was a full year before it was considered justifiable to attempt any dilatation of the stricture. This brings the case up to the point when I saw her for her present treatment.

The x-ray film of her chest showed a large pouch at the upper end of the esophagus, the apex of which was at about the arch of the aorta (Fig. 7). The esophagus narrowed to a tube about the size of ordinary macaroni, that is, it was between 3 and 2 mm. in width. The fundus of the stomach had been pulled into the chest half way to the arch of the aorta.

Under the fluoroscope very little barium passed through the esophagus and only after a long time.

An attempt with the flexible metal tip bougie to find the opening of the esophagus by the use of the fluoroscope was unsuccessful.

An examination under ether showed a large pouch which fitted over the vertical column like a horseshoe. In the left compartment of this shoe, guarded by a concentric web, the opening of the esophagus was found. This was about 2 mm. in diameter. A small bougie passed into this but did not reach the stomach.

An attempt was made to pass a bougie from the stomach into the esophagus, first under fluoroscopic control and then under the guidance of a small bronchoscope. This procedure was unsuccessful.

The patient was given ether a second time, and during this examination it was felt that a small bougie was passed into the stomach. The patient was transferred to the fluoroscopic room, hoping to locate the bougie in the stomach and bring the end out through

Fig. 7. X-ray film of the chest of a girl of twenty, who has had a stricture of the esophagus since she was two years old. There is a large pouch at the upper end of the esophagus. The fundus of the stomach has been pulled into the chest a third of the distance to the arch of the aorta.

the gastric fistula and attach a thread to it. This was unsuccessful. A number of attempts to have the patient swallow a string also were unsuccessful. Finally, however, a string did pass to the stomach. From this time on—some two or three months—the patient has been on retrograde bouginage, and a No. 28 French bougie now passes the stricture of the esophagus.

At the first ether examination, a circular stricture surrounding the pharynx was found about an inch above the arytenoids. The stricture is about one-fourth of an inch thick. There is no reason, of course, why a caustic should not cause a stricture at this point as well as at any other. I have, however, never happened to see one in this locality in my caustic cases. Nothing has been done to this stricture on account of its large lumen and because there is plenty of trouble below it.

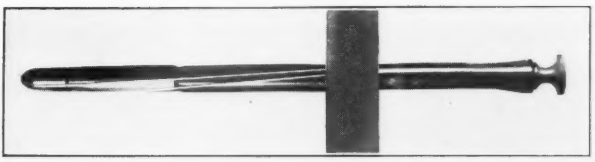


Fig. 8. The illustration shows a bougie fitted with a metallic tip carrying a detachable end. The detachable end is threaded and is held in place by a wire the tip of which is threaded. By turning the button at the end of the bougie the end of the bougie can be detached and left in any desired place in the esophagus.

The pouch at the upper end of the esophagus in this case has not yet shrunk as did the pouch in the high school boy. He, however, had had this condition only about five months, whereas this young lady had had hers probably for years. In her case the final management of the pouch is still an unsettled question.

FIBROSIS OF THE ESOPHAGUS.

It has been shown of late that the esophagus is subject not only to chronic infection but to acute infection as well; for instance, in pneumonia and in blood stream infections. It has been shown that chronic infection at the lower end of the esophagus often accompanies disease of the gall bladder or the liver. I have gone on record as stating that my belief is that the fundamental cause of cardiospasm is a fibrosis from infection of the lower end of the esophagus. In addition, there is also a twist of the terminal portion. My feeling is that this twist can come fairly early in the course of the disease. I should like to state that fibrosis of the esophagus as extremely common in such degenerative diseases as arteriosclerosis. Fibrosis shows itself either in an increase in the connective tissue under the muscularis mucosa, about the circular and longitudinal muscular layers, or in the two muscular layers themselves. If only one muscular layer is fibrosed, it is generally the circular.

The first photomicrograph shows a normal amount of connective tissue. This is seen to be fairly abundant below the muscularis mucosa and above the circular layer. There is a small amount also between the circular layer and the longitudinal layer, and then outside of the esophagus there is an abundant layer of connective tissue about as thick as the layer underneath the muscularis mucosa.

The second photomicrograph is from a case of chronic cholecystitis and cholelithiasis and icterus. The section shows a great increase of connective tissue. The subepithelial connective tissue is increased

in amount and is edematous. The subepithelial vessels are enlarged. The circular muscular layer is almost obliterated by fibrous tissue.

Other sections show enlarged and infected gland ducts. All the sections show chronic infection.

This specimen is a striking example of fibrosis of the terminal portion of the esophagus associated with chronic infection of the gall bladder.

826 BEACON STREET.

CII.

DIFFUSE SPASM OF THE LOWER PART OF THE ESOPHAGUS.*

HERMAN J. MOERSCH, M. D.,

JOHN D. CAMP, M. D.,

ROCHESTER, MINN.

Esophageal obstruction of neurogenic origin constitutes one of the most interesting phases of esophageal disease. Much has been written regarding this interesting problem, and although considerable progress has been made in dealing with it, there remains a very fertile field for further investigative effort. Localized spasm of the esophagus, cardiospasm and Bársony diverticula constitute the better known of these conditions.

Recently we have observed a small group of patients who undoubtedly belong to the category mentioned; who presented themselves because of intermittent dysphagia, and who, on roentgenoscopic examination, were found to suffer from diffuse spasm of the lower third of the esophagus. Although the condition in these cases closely simulated other types of esophageal obstruction of neurogenic origin, certain distinctive differences were readily apparent. Although the condition undoubtedly occurs with some frequency, review of the literature failed to elicit a reference to this interesting entity; it undoubtedly is classified with cardiospasm or other neurogenic changes.

The patients, eight in number, were equally divided as to sex, and were between fifty and seventy years of age. The clinical history, although suggestive of cardiospasm, more closely simulated the condition described by Teschendorf[1] as localized spasm of the esophagus. Dysphagia and pain constituted the outstanding symptoms. The dysphagia was usually intermittent, and was characterized by the fact that the point of obstruction frequently was situated at a higher level than in cardiospasm. Considerable variation existed as to the type of food or liquid that induced dysphagia, and the ability constantly to cause discomfort.

*Read before the seventeenth annual meeting of the American Bronchoscopic Society, Cleveland, June 11, 1934.

Pain was frequently a very pronounced symptom, and was the primary cause of the patient's seeking medical attention. It varied from a sense of dull discomfort under the lower half of the sternum to a severe, colic-like pain which might extend through into the back, up into the neck, or even out into the shoulder. In two of our cases the character and extension of the pain was such that a previous diagnosis of angina pectoris had been made. Careful investigation and the subsequent course of events indicated that such a diagnosis was incorrect, and that the pain was directly related to the diffuse spasm of the lower part of the esophagus. The pain associated with diffuse spasm of the lower part of the esophagus is usually induced by deglutition, and especially by the same type of food or liquid that produces dysphagia. Between attacks, the same substances may be ingested with impunity. Attacks of pain may also occur spontaneously, and waken the patient from a sound sleep, reaching such an intensity that narcotics may be required. The patients were all of a high-strung or temperamental make-up, and noted that spells of difficulty were frequently precipitated by increased nervous tension and overexcitement. Anxiety usually was very pronounced during an attack of dysphagia and especially when associated with pain. The duration of symptoms varied from two months to fifty years. Although general physical examination failed to reveal findings of characteristic importance, it was found advisable to make a thorough search for disease of the gastro-intestinal tract in every case, as will be referred to later.

The roentgenologic changes associated with the condition under consideration are particularly significant because of their value in differential diagnosis. Because of the changing character of the spastic defect it is best observed roentgenoscopically, since roentgenograms will record only certain phases of the rapidly changing image. In general, three types of spastic phenomena may be observed: (1) Diffuse, irregular spasm of the lower half or third of the esophagus; (2) diffuse narrowing of the lower half or third of the esophagus, suggesting tetanic spasm, and (3) multiple spastic segments of concentric narrowing. Each type may vary considerably in degree in the same and different patients, and at intervals during the examination the various types of spasm may occur in the same individual. The most frequent form is a diffuse, irregular spasm of the lower half or third of the esophagus (Fig. 1). It is obvious as soon as the first swallow of barium reaches the affected portion, and the spastic nature of the deformity is readily revealed by the rapid changes in the size of the lumen, due to the irregular peristaltic phenomena. The barium moves up and

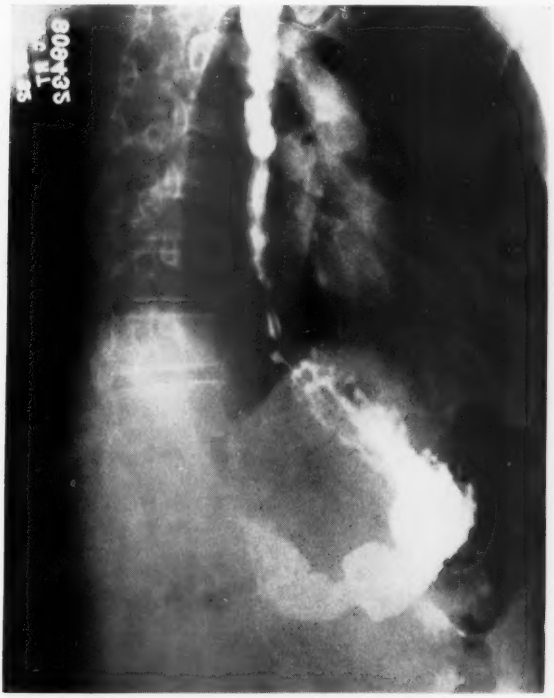


Fig. 1. Diffuse irregular spasm of the lower half of the esophagus.

down with peristaltic activity, thus revealing a flexible and intact esophageal lumen. At intervals the lower end of the esophagus will dilate slightly, allowing the content to enter the stomach, and the rapid changes in size of the lumen in this region will serve to distinguish the deformity from the fixed defect of cardiospasm. In some cases the diffuse, irregular spasm will be replaced at intervals by a temporary, diffuse, ribbon-like narrowing that has all the appearance of a tetanic contraction (Fig. 2). This, while it persists, simulates the diffuse narrowing of a benign stricture, with its partially filled esophagus below the stricture. In other instances the spasm may manifest itself as multiple regions of regular, concentric narrowing, between which the unaffected portions assume the appearance of diverticula (Fig. 3). Bársony and Polgár[2] have called attention to this peculiar neurogenic phenomenon and termed it "functional esophageal diverticulum." These transient, false diverticula may be mistaken for true diverticula and even esophageal ulcers, if the patient is not studied roentgenoscopically.

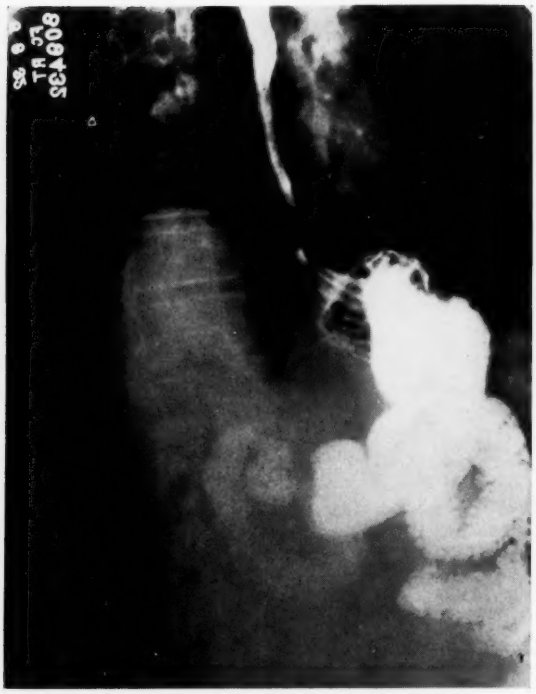


Fig. 2. Diffuse narrowing of the lower half of the esophagus, suggesting tetanic spasm.

A striking feature of this condition, which serves to distinguish it from typical cardiospasm, is the small degree of dilatation occurring above the affected portion, regardless of the duration of symptoms. In many cases of cardiospasm, following dilatation, there may be temporarily irregular diffuse spasm in the lower part of the esophagus, but this is not likely to be confused with the condition under consideration if the roentgenologic appearance before dilatation is kept in mind.

Intrinsic disease of the esophagus, such as ulcer or malignancy, may produce diffuse spasm simulating the condition we have noted, but the defect of the primary disease will serve to clarify the diagnosis.

Esophagoscopic examination reveals the lower end of the esophagus, corresponding to the spastic portion demonstrated roentgenoscopically, to be spastic, and the normal movements of the esophageal wall during respiration to be absent. In one patient a small, super-

Fig. 3. Multiple regions of regular concentric narrowing.

ficial area of ulceration was noted just above the cardia. Application of silver nitrate to this area produced pain, which was referred to the right shoulder. However, this did not influence the course of the symptoms. Passage of sounds over a previously swallowed silk thread failed to reveal anything further than a sense of spasticity of the lower part of the esophagus which required slightly greater force to overcome than is required in passage of sounds in the normal esophagus. Instrumentation of some patients caused pain, of such intensity that sedatives were required.

The most adequate treatment of diffuse spasm of the esophagus is still uncertain. In the past it has been treated in essentially the same manner as cardiospasm, by means of sounds and the hydrostatic dilator. Although improvement has been effected, it is in no respect as striking and satisfactory as in cardiospasm. In two of the cases of diffuse spasm it was impossible to pass sounds without production of excruciating colic. It was necessary to hospitalize the patients and

to give them large doses of codein and pentobarbital sodium for relief of pain. A Rehfuss tube was inserted carefully into the stomach and all food was given through the tube over a period of a week, permitting the esophagus to be at rest. Only at the end of this period was it possible to carry out dilations and then with indifferent success.

Although the exact etiologic basis of diffuse spasm of the lower part of the esophagus is not definitely known, several factors offer fertile fields for conjecture. The complexity of the problem, however, is readily apparent from a survey of the experimental physiology dealing with the esophagus, and the divergence of opinion as to the exact function of its component parts. The influence of nervous instability is very striking, as previously has been mentioned. This is somewhat in contradistinction to cardiospasm, in which in our experience nervous instability is a minor element. Teschendorf, likewise, in commenting on localized spasm of the esophagus, stated that it is more likely to afflict young, neurotic individuals than others. Lust[3] also has observed that it may afflict children who object to certain types of food.

We are inclined to believe that intra-abdominal pathologic conditions of highly nervous individuals may act as causative factors. However, sufficient evidence is not at hand to substantiate such an hypothesis. Because of this belief, we feel it is important that careful study of the gastro-intestinal tract, gall bladder and genito-urinary tract be made in every case. The following case is illustrative of the difference between cardiospasm and diffuse spasm of the esophagus, and the possible relationship of the latter to intra-abdominal pathologic conditions.

REPORT OF CASE.

CASE 1.—A man, 53 years of age, was first seen at The Mayo Clinic in November, 1929. He always had enjoyed the best of health until the onset in 1924 of the illness for which he came to us. At that time, he had noted regurgitation of mucus following meals. Dysphagia, with stoppage of food under the lower part of the sternum, followed by regurgitation, soon had developed, solid food and cold liquids especially had produced difficulty. The symptoms gradually had increased, and, during two years, the patient had lost 47 pounds (21.3 kg.). In 1926 a diagnosis of carcinoma of the lower part of the esophagus had been made, and gastrostomy advised. This had been performed, the surgeon confirming the previous diagnosis. The patient gradually had failed in strength and weight during the next year. In June, 1927, radium had been applied to the lower part of the esophagus. Following the second application, a month after the first treatment, the patient had suffered from severe hematemesis necessitating transfusion. In the following year the patient had been able intermittently to swallow small amounts of liquid, but otherwise his condition had remained unchanged. In 1928, following roentgenologic review of the esophagus, a diagnosis

of cardiospasm had been made. Treatment by passage of bougies had been instituted, with little improvement. Because of the disagreement in diagnosis and the patient's failure to improve following treatment, the family suspected the difficulty might be due to a type of insanity and proceedings had been started to commit him to an asylum for the insane. This attempt, however, had been unsuccessful.

When the patient came under our care he was able to swallow only liquids by mouth, and these slowly. All other nourishment was administered through the gastrostomy tube. The patient was very much emaciated and depressed, and weighed 124 pounds (56.2 kg.) as compared to 207 pounds (94 kg.), his normal weight. Physical examination gave essentially negative results. Examinations of urine, blood counts, the Wassermann reaction of the blood and roentgenograms of the thorax gave negative results. Roentgenoscopic examination of the esophagus, however, revealed the presence of cardiospasm, with diffuse dilatation. In spite of the marked obstruction, the patient was able to swallow a thread without difficulty. Dilation was instituted by means of Plummer sounds, and was carried to the diameter of a number 45 French sound. Two days later, the esophagus was dilated with a hydrostatic dilator under water pressure of 22 feet (670.5 cm.). Following the dilation, the patient was able to eat everything without difficulty; the gastrostomy tube was removed and the wound was allowed to heal. Symptoms were entirely absent for a year and a half. Then gradual recurrence of symptoms, together with discomfort through the thorax, appeared.

The symptoms gradually increased, and the man returned to the clinic in January, 1933, at which time his esophagus was almost completely obstructed. His weight was now 141 pounds (64 kg.), a drop of 19 pounds (8.6 kg.) during the preceding six months. Roentgenoscopic examination revealed change in the roentgenologic picture seen at the former examination. There was now diffuse spasm of the lower third of the esophagus. Dilation up to the diameter of a number 60 French sound now failed to give the patient relief, but rather increased the substernal discomfort. The possibility of an associated gastro-intestinal lesion was suspected, and a second fluoroscopic examination of the esophagus and stomach was attempted following dilation. The esophageal picture remained unchanged, and due to the marked spasm, it was impossible to get sufficient barium into the stomach to allow it to be adequately visualized. Pentobarbital sodium, grain 1½ (0.1 gm.) was given twice a day, and diathermy was administered over the lower end of the sternum daily for a week, in order to try to cause relaxation of the spasm of the esophagus, as we have reported elsewhere. By the end of a week, the patient was able to swallow liquids with a fair degree of success. Re-examination, although it still revealed spasm of the lower part of the esophagus, disclosed that there was sufficient relaxation to permit the barium to enter the stomach, and an obstructive lesion was demonstrated at the pylorus. Careful review of the patient's history failed to elicit anything to suggest a possible ulcer. Operation for the obstructing lesion at the pylorus was advised and performed. A large, perforating duodenal ulcer was found, repaired, and gastro-enterostomy performed. The patient made rapid recovery and has been able to eat since without the least difficulty.

COMMENT.

European investigators, especially, have stressed the importance of neurogenic changes in the esophagus and have emphasized its

association with disease elsewhere in the gastro-intestinal tract. Such bases for cardiospasm have been suggested by Teschendorf, Strauss,[4] Schlesinger,[5] Meyer,[6] Soper and Cassidy[7] and others. Einhorn and Scholz[8] stated: "Spasm of the esophagus is seen in disease of the esophagus itself, and organic disease of the upper part of the stomach and acute disease of the gall bladder." Lookwood,[9] Budde,[10] Bársony[11] and others have reported cases in which cardiospasm has been definitely relieved by surgical treatment of an associated intra-abdominal lesion. Such a radical procedure, however, hardly seems justified unless the intra-abdominal disease is of itself sufficient to indicate the need for operation. Further support for such a theory is offered by the experimental work of Cannon,[12] Meltzer,[13] Carlson and Luckhardt,[14] Hanzlik and Butt,[15] and others who definitely have demonstrated that changes involving the vagus nerves readily cause alterations in the activity of the esophagus. The experimental observations of Carlson and his coworkers[16] that stimulation of the gall bladder, urinary bladder and intestines will lead to esophageal activity is especially worthy of notice. The exact importance and influence of the sympathetic nerves remain, however, debatable.

The work of Mosher[17] on the involvement of the esophagus in acute and chronic infection, likewise offers a possible explanation of the cause of diffuse spasm of the lower part of the esophagus. Intra-abdominal infections such as those encountered in cholecystitis, appendicitis and duodenal ulcer may be carried by lymphatic vessels to the esophagus and give rise to localized esophageal infection; thus diffuse spasm may be produced. Which explanation is correct is difficult to say, and considerable investigation will be required before the cause is known.

In conclusion, it is our desire to call attention to an interesting clinical entity, characterized by intermittent dysphagia and substernal pain. On roentgenoscopic examination diffuse spasm of the lower part of the esophagus is disclosed. The etiology is not exactly known.

THE MAYO CLINIC.

BIBLIOGRAPHY.

1. Teschendorf, W.: Die Röntgenuntersuchung der Speiseröhre. Ergebn. d. med. Strahlenforsch., 3:175-288, 1928.

2. Bársony, Theodor, and Polgár, Franz: Symptomlose und funktionelle Speiseröhrendivertikel. Fortchr. a. d. Geb. d. Röntgenstrahlen, 36:593-602, 1927.

3. Lust, F.: Zur Klinik des Oesophagospasmus. Monatschr. f. Kinderh., 27: 9-18 (Oct.), 1923.

4. Strauss: Röntgenbeobachtungen über funktionelles Verhalten der Speiseröhre. Berl. klin. Wchnschr., 54:709 (July 16), 1917.

5. Schlesinger, Hermann: Bisher unbekannte viszero-motorische Reflexe des Verdauungstraktes und ihre Bedeutung für die Diagnostik. Mitt. a. d. Grenzgeb. d. Med. u. Chir., 38:8-15, 1924.

6. Meyer, Hermann: Entstehung und Behandlung der Speiseröhrenerweiterungen und des Cardiospasmus. Mitt. a. d. Grenzgeb. d. Med. u Chir., 34:484-513, 1922.

7. Soper, H. W., and Cassidy, L. D.: Cardiospasm with Special Reference to Etiology. Am. J. Med. Sc., 177:386-389 (Mar.), 1929.

8. Einhorn, Max, and Scholz, Thomas: Über Spasmen des Verdauungskanals und deren Diagnose. Arch. f. Verdauungskr., 37:1-16, 1926.

9. Lookwood: Quoted by Meyer.

10. Budde, Werner: Über Cardio- und Ösophagospasmus bei Ulcus ventriculi. Mitt. a. d. Grenzgeb. d. Med. u. Chir., 38:525-537, 1925.

11. Bársony, Theodor: Funktionelle Speiseröhrendivertikel (Relaxations-Divertikel). Wien. klin. Wchnschr., 39:1363-1364 (Nov. 18), 1926.

12. Cannon, W. B.: Esophageal Peristalsis After Bilateral Vagotomy. Am. J. Physiol., 19:436-444 (Aug.), 1907.

13. Meltzer, S. J.: On the Causes of the Orderly Progress of the Peristaltic Movements in the Esophagus. Am. J. Physiol., 2:266-272 (Mar.), 1898.

14. Carlson, A. J., and Luckhardt, A. B.: Studies on the Visceral Sensory Nervous System. X. The Vagus Control of the Esophagus. Am. J. Physiol., 57: 299-335 (Sept.), 1921.

15. Hanzlik, P. J., and Butt, E. M.: Reactions of the Crop (esophageal) Muscles Under Tension, with a Consideration of the Anatomical Arrangement, Innervation and Other Factors. Am. J. Physiol., 85:271-289 (June), 1928.

16. Carlson, A. J., Boyd, T. E., and Pearcy, J. F.: Studies on the Visceral Sensory Nervous System. XIV. The Reflex Control of the Cardia and Lower Esophagus in Mammals. Arch. Int. Med., 30:409-433 (Oct.), 1922.

17. Mosher, H. P.: Involvement of the Esophagus in Acute and in Chronic Infection. Arch. Otolaryng., 18:562-598 (Nov.), 1933.

CIII.

THE CLINICAL DIAGNOSIS OF PRIMARY CANCER OF THE LUNG.*

MILLARD F. ARBUCKLE, M. D.,

ST. LOUIS.

As you know, great advances have been made in the field of thoracic surgery in the past few years.[1] One of particular note has to do with the method of dealing with malignant disease of the lung and, because of our interest in this subject as bronchoscopists, it was thought worth while to present for your consideration a discussion of the clinical diagnosis of primary cancer of the lung.

It has been shown and is now generally conceded by those best entitled to an opinion, that after all other methods of study have been carried out, the final evidence upon which the diagnosis must rest,[2] if it is to be made early enough in the course of the disease to permit of successful treatment, must be produced through the efforts of the bronchoscopist—i. e., biopsy taken bronchoscopically.

For purposes of discussion, several classifications of the disease may be used, such as early or late; hilar or parenchymal; squamous, adenocarcinomal, "oat-cell," or a combination of these, according to cellular origin.[3]

At this time I shall speak of the findings in both the early and late stages of the disease. There is, of course, a vast difference in the two pictures. A point of practical importance is that in the first we may offer hope of successful treatment, while this is not true of the later stages of the disease. The significance of this statement is accentuated by the fact that we are now able in a large percentage of cases to establish the diagnosis by microscopic study while the neoplasm is still confined to the lumen of the bronchus. In the late stages of the disease, bronchoscopic examination usually is not required in order to establish the diagnosis. In many cases seen by this time the pleural cavity is filled with fluid containing malignant cells and

---

*From the department of Otolaryngology, Department of Radiology and the Chest Service, Washington University School of Medicine, St. Louis, Mo.

Read before the seventeenth annual meeting of the American Bronchoscopic Society, Cleveland, June 11, 1934.

the picture is so clear that the diagnosis is evident to any competent observer.

In view of recent developments in thoracic surgery, we feel that a great stride forward has been taken in the management of cancer of the lung. Since all other methods of treatment have been found wanting and because of the prospect held out by this method, we think our zeal in urging early and competent examination is justified. It is true that even among the more enlightened and so-called "best circles" of medical practitioners, the early symptoms of this disease frequently are overlooked or charged to some other cause. The statement that early symptoms are "overlooked" or "not recognized" is made advisedly. The reason is, of course, that the most prominent symptom, cough, usually is explained on some other basis. The relative inaccessibility of the site of the disease is, too, in a measure responsible for the fact that correct diagnosis has so often been missed. Furthermore, in our early training we have not been taught to associate cough with malignant disease of the lung and, in addition, we have been led to believe that the disease is rare. In this connection, Graham calls attention to the fact that "Textbooks of both pathology and medicine published less than a decade ago state this condition constitutes only about 1 per cent of all carcinomas."[4] Whereas, we now know that carcinoma of the lung constitutes 10 to 12 per cent instead of six-tenths to one and five-tenths per cent of all cancer.[5]

The knowledge that cancer of the lung comprises such an important percentage of all cancer has led to a definite feeling in the minds of some that carcinoma of the lung is on the increase. Every writer on this subject comments on the fact that diagnosis has been made in the past either in the postmortem room or in the terminal stages of the disease, and there is evidence that carcinoma of the bronchus has often been overlooked at autopsy because of the insignificant part it played as compared with that of secondary involvement, namely, abscess formation, the pathologic diagnosis being put down as "death from abscess of the lung" rather than "carcinoma of the lung, with abscess formation." It is certain that the diagnosis is now being made more frequently, but I am not sure that the environment of our type of civilization may not be an etiologic factor in accounting for an increase in incidence. This thought is supported by a recent conversation with a practitioner, who has worked for the past several years in Alaska, and who made the statement that cancer was practically unknown among the natives, and that he felt that their way of living was the reason for this. Again, it is generally known that cancer of the esophagus is a common disease among the males in China, while

it is rare among the females. This was so strikingly demonstrated to him that Charles Mayo, I am told, expressed the belief that this might be because the men drink their tea scalding hot, while the women, who are not served until the men have finished, habitually drink it when it is much cooler. The facts concerning the incidence of this disease have also been corroborated by Dr. C. S. Jung, most brilliant Chinese physician.

As evidence of a change of feeling on the part of those dealing with malignant disease is the statement of Graham, that when a man of forty-five or thereabouts presents himself with unexplained atelectasis of one or more lobes, the diagnosis should be considered cancer of the lung until proven otherwise.

In attempting to establish the basis for an early diagnosis, we understand and share the feeling of Meakins and MacLeod when they write: "The majority of the clinical descriptions of carcinoma of the bronchus leave us in a hopeless state of mind in regard to its early recognition."

To add to the difficulty, most of the typical signs and symptoms are those which may accompany other pulmonary disease; and, in the late stages of the disease metastasis and secondary changes help further to obscure the diagnosis. It has been found that this disease may occur much earlier in life than we formerly thought, our youngest case being aged twenty-seven years.

In our experience, teamwork in studying these cases has produced best results; in every case the findings have been the result of the joint efforts of the internist, the radiologist, the thoracic surgeon, the bronchoscopist and the clinical pathologist.

In a series of twenty-one cases of endobronchial cancer studied with Graham and Singer, the tumor was visible bronchoscopically in twelve, while in nine it was not seen. Of the twelve cases the tumor was located in the right main bronchus in nine; in the left main and upper lobe in two; and in the trachea in one. In those in which the tumor was not seen the location was in the parenchyma, four on the right and five on the left side. A few typical cases are herewith presented.

In forty-one cases seen in this service, in which no bronchoscopy was done but in which the diagnosis was definitely established at autopsy, the location of the tumor was as follows: Right bronchus, eleven; right lung, four; right periphery, three; left bronchus, nine; left lung, six; right hilar region, two; peripheral without bronchial connection, one; parenchymal, one; both lungs, one; and three not

stated. In many cases the disease was in the late stages, presenting marked secondary changes, such as abscess formation, empyema, cachexia and other signs of late stages of carcinoma. The diagnosis in many of these cases was established before death by a study of the cells in the fluid of the pleural cavity. While bronchoscopic examination was not made in this group of cases, it is evident from the location of the tumor that had these patients been seen early in the course of the disease the diagnosis in many could no doubt have been established bronchoscopically.

Symptoms early in the course of primary cancer of the lung are very few, and they are those which we would expect in the presence of any irritative, destructive lesion of the bronchial mucous membrane, namely, cough, expectoration, discomfort or pain. Later on, there is a bronchial obstruction, abscess formation, much increase in sputum, hemoptysis, pain, dyspnea, sepsis, anemia and the other findings which go with lung abscess. Bleeding from the lesion, in our experience, has been comparatively rare, and then late. It follows ulceration and the opening of blood vessels. The amount of blood lost by hemoptysis usually is not of great import, but the occasional case has been seen in which, judging from the amount of blood lost, a fairly large vessel has been opened by erosion.

The cough of bronchogenic carcinoma usually is the first symptom and may be the only one over a long period. It is peculiar in that it is persistent, racking, paroxysmal in nature, unexplained and nonproductive. It is a well established fact that cancer of the bronchus may be present for a long time, even months or years, with practically the only symptom being that of cough.

Probably the second symptom to appear is that of a feeling of uneasiness or discomfort, in the region of the lesion. This sensation gradually increases in severity until late in the disease when it becomes pain, its most characteristic feature being its persistency from the first. Its severity usually is sufficient to require narcotics for relief. We have been impressed with the frequency with which patients with a cancer of the lung give a history of an injury to the chest wall such as a severe blow without laceration.

As the lesion encroaches upon the lumen of the bronchus, the patient may develop an asthmatoid wheeze, the result of a partial obstruction of the bronchus, similar in a way to that described by Jackson,[6] years ago, in his book on the diagnosis of foreign body in the bronchus.

Radiopaque oil introduced into the bronchus will demonstrate irregularities or tumefaction in its wall.[7] The use of iodized oil in the

study of pulmonary disorders has proven of the greatest help in localizing and visualizing radiologically the site of such lesions. In some cases it is possible to inject lipiodol satisfactorily only with the aid of the bronchoscope. We find the most satisfactory plan is to place a rubber catheter through the bronchoscope into the bronchus to be injected, then withdraw the bronchoscope, inject the oil and make the plate immediately.

The man who specializes in disease of the chest has certain niceties of practice not usually carried out by the man doing a big general practice. These include procedures such as special posing for plates to demonstrate fluid levels, if present, and to direct the flow of radiopaque oil; also special methods of introducing the radiopaque oil.[8]

It is, of course, unusual to see carcinoma of the lung before bronchial obstruction has occurred, but the fact remains that if and when such cases are seen, it will be because the early symptoms lead us to suspect the presence of cancer, and the only method of establishing a diagnosis is by biopsy.

Formerly the diagnosis, when made during life, was usually reached by a process of exclusion. That is to say, all other disease affecting the pulmonary tract having been ruled out, carcinoma was put down as the only remaining possibility. Great stress was laid on the finger-like projections seen in the x-ray plates and on certain other visible changes. As late as 1933, the Yearbook of Radiology contains an article in which it is erroneously pointed out that such changes are cancerous in nature and profess to show improvement as a result of roentgen radiation. Pancoast of Philadelphia long ago pointed out that these changes are secondary and are chiefly inflammatory rather than malignant in nature, resulting at first from broncial obstruction with atelectasis infection and abscess formation. As the tumor grows in size, it does, of course, form solid metastatic masses which will cast a shadow, but in cases such as these it is utter folly with our present knowledge to think for a single moment of securing lasting results with any form of radiation now available.

With a consideration of the history and the findings as shown in the physical and radiologic examination, we may know that we have to deal with an obstructive lesion. By exclusion and deduction, we are enabled to make a tentative diagnosis of bronchogenic carcinoma and we are in a position to tell the patient we feel beyond question of doubt that the diagnosis in his case is bronchogenic carcinoma. When faced with such a situation we are in duty bound to recom-

mend not only biopsy but in addition a careful visual examination of the involved area with special regard as to the extent and proximal distribution of the lesion. Meakins, Jackson, Iglauer and other writers have expressed a similar feeling, and Carlson and Ballon, in discussing bronchoscopy, say, "It is the only method we possess of making the diagnosis early."[9] Of course, we share in this feeling.

It is true that some able internists still are not enthusiastic about having patients bronchoscoped for diagnostic purposes. This reluctance is based on the fear of harm to the patient and also on a failure to realize the advantages of direct inspection and biopsy. This feeling usually disappears with experience.

I do not agree with those who teach that bronchoscopy is a "perfectly simple matter," or that it is entirely free of danger. We are forced to rely on monocular vision, to view the field from one direction only and this with artificial light; reflections and distortions, not to speak of the more or less constant movements of respiration and coughing, add to the difficulty. Occasionally the bronchoscopist is called upon to face unexpected and serious difficulty such as hemorrhage and to supply the remedy.

Recently I have had an experience which causes me to make the following comment: On May 16, 1934, I did a bronchoscopy on a patient suspected of having carcinoma of the lung with abscess, with the intention of making a biopsy. Biopsy was not made, because of the great amount of purulent secretion and a slight bleeding from granulation tissue and because the patient was slightly cyanotic. We were content to apply suction to the abscess cavity and put in oil, having in mind the necessity for further examination. This patient had had three doses of hyoscin at forty-five minute intervals. With the first dose of hyoscin he was given one-fourth grain of morphin. Thirty minutes after the third dose of hyoscin he received an additional one-sixth grain of morphin as pre-operative medication, but he apparently had completely revived from the effects of these drugs, since he sat up and talked freely with members of the staff some four or five hours later. However, about nine hours later the patient died suddenly. Autopsy was not permitted, but we think he had a brain embolus. Since it is well known that brain embolus is one of the most common and most feared complications of lung disease, it is only fair to state that this is always a possibility in dealing with such cases, bronchoscopically or otherwise.

In one of the accompanying case reports, the matter of severe hemorrhage at the time of operation is noted.

In some cases it is necessary to make repeated bronchoscopic examinations in order to establish the location and extent as well as the nature of the lesion as in case No. 1 of this series. As in the larynx, repeated biopsy may be and often is necessary. The old question of the advisability of making biopsy may be raised. The reply should be that it is the only method by which the diagnosis can be definitely established at a time when it is worth while to the patient. In making the biopsy great care should be exercised to preserve the barriers already provided by Nature between the seat of the disease and the mediastinum—i. e., the bronchial wall. Of immediate importance is the possible danger of causing a pneumothorax or empyema if the bronchial wall is cut through. Furthermore, careful bronchoscopic examination and study will help materially in outlining the treatment to be administered—i. e., the exact location and extent, proximally at any rate, of a given lesion. This will be an important factor in considering its operability and the extent to which this procedure may be applied.

Apparently there is a very considerable difference in the course of the disease, depending upon the location of the lesion, whether parenchymal or hilar, and upon the type of cell of which the tumor is made up. Bronchogenic carcinoma when in the larger tubes, may remain localized and free of metastasis for a comparatively long period. It is surrounded by a wall of cartilage, similar to that which is true of cancer of the vocal cord, and dense fibrous tissue, at least until it breaks through. When located in the parenchyma there are no such barriers to prevent its spread and the course is much more rapid.

REPORT OF CASES.

CASE 1.—J. L. G., male, aged 48, physician, whose case report has been published elsewhere,[4] was first seen in the Chest Service of Barnes Hospital by Dr. Evarts Graham and Dr. J. J. Singer on February 27, 1933. The report of their findings follows:

"Had had repeated attacks of cough and fever with pain in the left side of the chest for a period of seven months. Other complaints were loss of weight and general lassitude. In January, 1929, he had a pneumonia of the lower lobe of the right lung (the other lung). The pneumonia in the right lung was said to have spread and to have involved the entire lung. After several weeks, however, he stated that he recovered fully from the attack of pneumonia until his symptoms appeared insidiously in the left lung more than three years later.

"In July, 1932, he complained of malaise with chilly sensations and a temperature of 104°. At that time nothing was found on physical examination to explain his symptoms. The leucocytes numbered 17,000. August 11, 1932, a roentgen examination revealed a fan-shaped shadow with the base outward in the region of the left axilla. By August 20, his symptoms had subsided and the x-ray shadow had become smaller. October 7, 1932, he had a repetition of his former symptoms with a return of the former x-ray shadow. These symptoms subsided in a few days

but recurred again about October 20, 1932. At this time there was some dullness, and a diagnosis either of interlobar empyema or of lung abscess was made. When an attempt was made to aspirate pus, December 5, 1932, a pneumothorax developed, after which a marked improvement in his symptoms was noted, although there was a complaint of some pain in the left side of the chest. Artificial pneumothorax was then continued and the patient showed steady improvement until ten days before his admission to the Barnes Hospital (February 17, 1933), when he had a recurrence of fever and discomfort. At no time was there any actual pain and never any bloody sputum.

"The patient was of medium build with a suggestion of loss of weight and a rather pale complexion. The left side of the chest moved less than the right, and the breath sounds were diminished or absent on that side. A roentgen examination showed the left upper lobe to be atelectatic with pneumothorax present. The lower lobe seemed to be fully expanded and adherent to the chest wall. The blood examination showed 4,800,000 red cells, 11,500 leucocytes and 85 per cent hemoglobin. Because of the presence of the atelectasis of the upper lobe, and in view of the patient's history of an insidious onset, a diagnosis was made of an obstruction of the bronchus of the upper lobe, probably by a tumor. Bronchography with iodized oil substantiated the diagnosis of obstruction of the bronchus of the left upper lobe. A bronchoscopic examination was accordingly advised and performed by Dr. Arbuckle, March 1, 1933. At this time tissue was removed which seemed microscopically to be only granulation tissue. The patient's symptoms improved following this examination, because the obstruction of the bronchus had been somewhat relieved. A bronchoscopic examination was repeated on March 14, 1933, and again on March 21, 1933, and specimens were removed again at both examinations. Both of these specimens revealed squamous cell carcinoma of the bronchus. The patient was advised to have the upper left lobe removed because of the presence of the carcinoma obstructing the bronchus of that lobe.

"At the operation, however, which was performed April 5, 1933, with intratracheal anesthesia of nitrous oxide and oxygen, it was found that the carcinoma extended so closely to the bronchus of the lower lobe that it was impossible to save the latter bronchus. Moreover, there were many nodules in the upper portion of the lower lobe about which uncertainty existed as to whether they were tumor tissue or areas of inflammation. Finally, also the interlobar fissure was not complete. For all these reasons it was decided to remove the entire lung. The adhesions between the lower lobe, chest wall and diaphragm were separated without great difficulty. A small rubber catheter was tied tightly around the hilus as close to the trachea as possible. Crushing clamps were placed on the hilus below the catheter and the lung was cut off with an electric cautery knife. The open end of the left main bronchus was carefully cauterized with the actual cautery as far up as the catheter would permit in order to destroy the mucous membrane thoroughly. A transfixing double ligature of No. 2 chromic catgut was tied around the stump just distal to the catheter and the latter was then removed. No bleeding occurred. Another transfixing ligature of No. 2 chromic catgut was placed where the catheter had been. The stump of the pulmonary artery was then ligated separately with catgut, seven radon seeds of 1.5 millicuries each were inserted into various parts of the stump. Several enlarged tracheobronchial glands were removed from the mediastinum and seven ribs, from the third to the ninth, inclusive, were removed from the transverse processes of the spine to the anterior axillary line. The ribs were removed for the purpose of allowing the soft tissues of the chest wall to col-

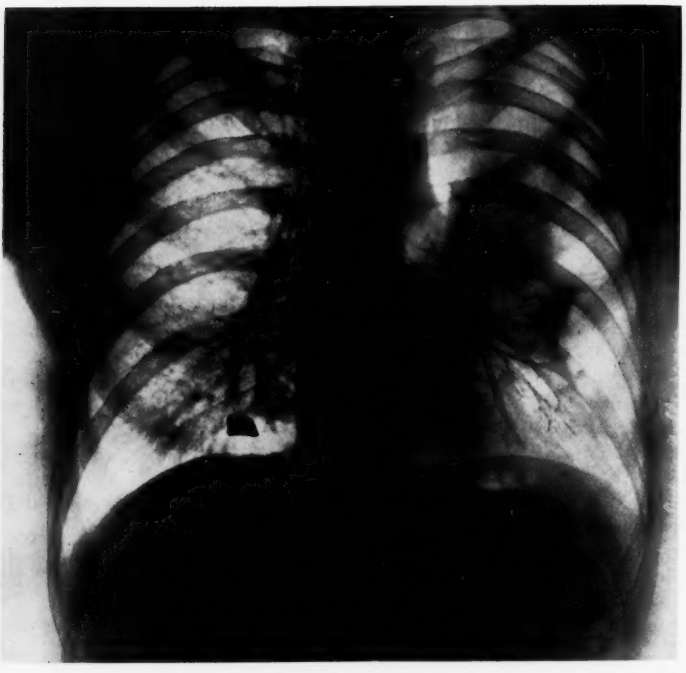


Fig. 1. Case 1. February 27, 1933. Postero-anterior and left anterior oblique view of chest, after instillation of lipiodol-bronchography.

In the postero-anterior view there is evidence of a pneumothorax pocket on the left, with compression of the left upper lobe, with air lying both along the mediastinal and lateral aspects of the lobe. Lung markings are seen throughout the lower portion of the lung field, to the lateral chest wall. The descending branches on the left are well filled with opaque material and appear to be within normal limits. The bifurcation of the left main bronchus is fairly well visualized and some opaque material outlining the ascending bronchus on the left is noted. The main ascending bronchial branch to the left upper lobe bronchus is distorted, narrowed, and filled only for a short distance. Only a small amount of opaque material is seen in the region of the right main bronchus. Left anterior oblique view adds nothing to the foregoing.

X-ray diagnosis: Pneumothorax, left. Indeterminate for bronchography.

lapse against the bronchial stump and therefore to obliterate as much as possible the pleural cavity. The first and second ribs were not removed at this time, merely because it was desired not to do too much operating at once. Nevertheless, it was felt that there would be some danger of the development of an empyema in the upper part of the pleural cavity because of the failure to obliterate that space. The wound was closed tightly, but provision for drainage was made by the use of an air-tight catheter brought out through a stab wound.

"The patient left the operating room in excellent condition, but was nevertheless given a transfusion of 500 cc. of blood. The closed drainage yielded about 800 cc. of serosanguinous fluid during each of the first two days. After that period

the drainage rapidly diminished and practically ceased on the fifth postoperative day. The catheter was gradually withdrawn. The wound healed by primary intention. . . . On the ninth postoperative day there was a collection of air and pus in the extreme upper portion of the pleural cavity. The rest of the pleural cavity was completely obliterated by what seemed to be solid healing of the soft tissues of the chest wall against the mediastinal pleura. . . . It was evident, both from the previous accumulation of air in the unobliterated portion of the cavity and also because the patient was coughing up pus that there was a small communication between the unobliterated portion of the pleural cavity and the bronchial stump. . . . The patient's temperature and pulse had been normal during the entire time of the drainage of the empyema cavity. May 22, 1933, through a posterior incision, the first and second ribs were removed in almost their entire length. There was almost no reaction after this operation. The small remnant of pleural cavity was completely obliterated at once.

"The pain in the back subsided and within three weeks the wounds were all solidly healed. The patient's strength gradually increased, his appetite was excellent and he was discharged from the hospital June 18, 1933, looking and feeling better than he had for many months previously. His only complaint was some dyspnea on exertion, but he had been walking about the hospital for two weeks before his discharge. His vital capacity on admission was 3,500 cc.; at his discharge it was 1,650 cc. . . . A report received from him, July 25, 1933, five weeks after his discharge, stated that he had gained eight pounds (3.6 kg.) at home, that he was able to walk about a mile without much dyspnea, that he was driving his automobile and that his strength was rapidly improving. The dyspnea was rapidly becoming less.

"On October 31, 1934, he is carrying on with his practice and seems to be in normal health."

CASE 2.—C. B., white, housewife, aged 44 years, entered Barnes Hospital August 9, 1931.

She stated that she had been fairly well up until one year previous to admission when, following a mild attack of hay fever, the cough which she had at that time persisted and she gradually began to feel weak.

At the onset the cough was quite productive, of a white, rather tenacious sputum, but had recently decreased in amount. There had been occasional streaks of blood in the sputum. Patient had lost about five pounds. She had suffered no pain, but had an occasional dull ache in the right chest.

The past history revealed that at the age of twenty, she had had a severe cough and on one occasion had coughed up blood. At that time she spent four years in Colorado, after which time the cough disappeared and her health became normal.

Family history was negative.

She had a panhystorectomy in 1927 and a thyroidectomy in 1931.

Physical examination revealed a well developed, well nourished white female. General examination was essentially negative, with the exception of the chest, where there was a slight increase in the whispered voice sounds over the right chest posteriorly from the third to the seventh rib.

There was paralysis of the right vocal cord, which the patient stated had been present since the thyroidectomy.

Laboratory findings were essentially normal.

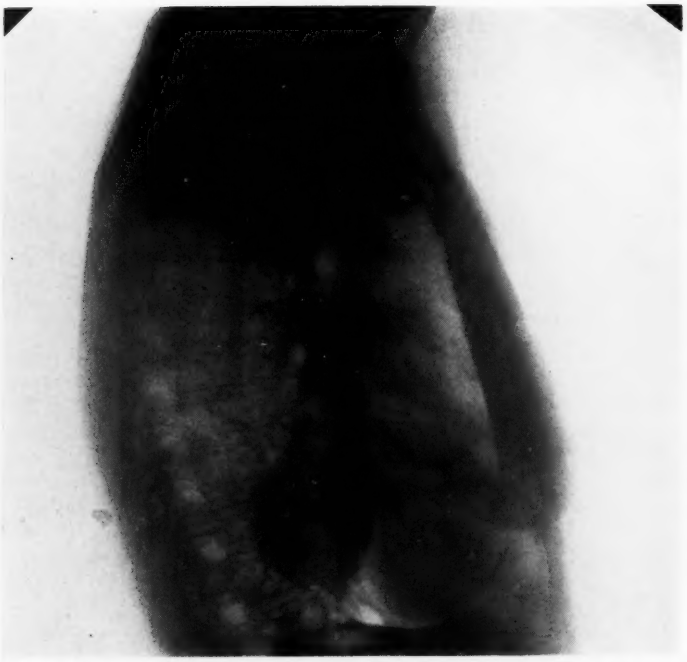


Fig. 2. Case 2. August, 1931. Anteroposterior and lateral views of chest.

Cardiac outline is within normal limits. There is a moderate increase in hilus on either side, with a slight increase in all bronchial markings, which extend far out. Extending beyond the right heart border, opposite the anterior ends of the fifth and sixth ribs on the right, is seen a rounded, well circumscribed area of increased density about 5 cm. in diameter.

In the lateral view this density is well visualized and lies in the posterior mediastinum.

X-ray diagnosis: Tumor of posterior mediastinum.

X-ray films revealed a well circumscribed area of increased density, about 5 cm. in diameter, extending beyond the right border to the anterior ends of the fifth and sixth ribs. Reported in legend accompanying plates.

The diagnosis at this time was tumor of the posterior mediastinum.

The patient entered the hospital the second time in August, 1932, at which time she stated that during the past year from her previous admission, she had lost weight and strength, and for almost twelve months had been confined to her bed. During the six months previous to her second admission she had noticed some dyspnea and occasional pain in the right chest. She had lost ten pounds.

Physical examination revealed an increase in the size of the tumor mass in the right lower chest and there were dullness and diminished breath sounds. She had occasional râles over the lower half of the entire right chest. Whispered voice sounds were high pitched. Lipiodol injected at this time showed an obstruction in the right main bronchus. The patient was bronchoscoped and a mass was found in the right main bronchus.

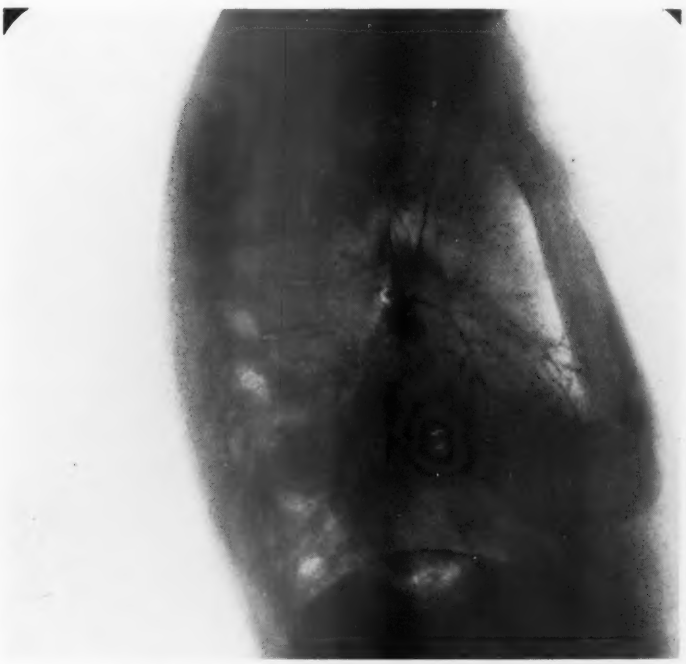


Fig. 3. Case 2. October 14, 1932. Anteroposterior view of chest—bronchography.

Lateral view of chest.

See previous films and report.

In the anteroposterior view a small amount of opaque material outlines the descending bronchial markings in the mid-portion of the right lung field. There is a collection of opaque material in the region of the bronchus to the right lower lobe which shows puddling. There is good filling of the bronchial branches in the mid-portion of the right lung field. There is evidence of obstruction of the bronchus to the right lower lobe, and puddling of the opaque material is seen in the region of this bronchus, opposite the seventh dorsal vertebra. It presents a fluid level along its superior border, and has an irregularity suggesting a filling defect within the bronchus. There is complete opacity over the lower third of the right lung field, and slight retraction of the cardiac shadow to the right.

X-ray diagnosis: Atelectasis, right lower lobe. Bronchial obstruction. (?) Tumor of lung.

Biopsy was taken, which on microscopic examination proved to be chronic inflammatory tissue. The patient was bronchoscoped on three other occasions and each time the biopsy failed to show carcinoma. The biopsy was very superficial, because when an attempt was made to do the biopsy there was extremely free bleeding which, as we suspected, came from beyond the tumor.

The patient was discharged from the hospital, the diagnosis being probable carcinoma of the lung, unverified. She was given two courses of deep x-ray therapy over the anterior and posterior right chest.

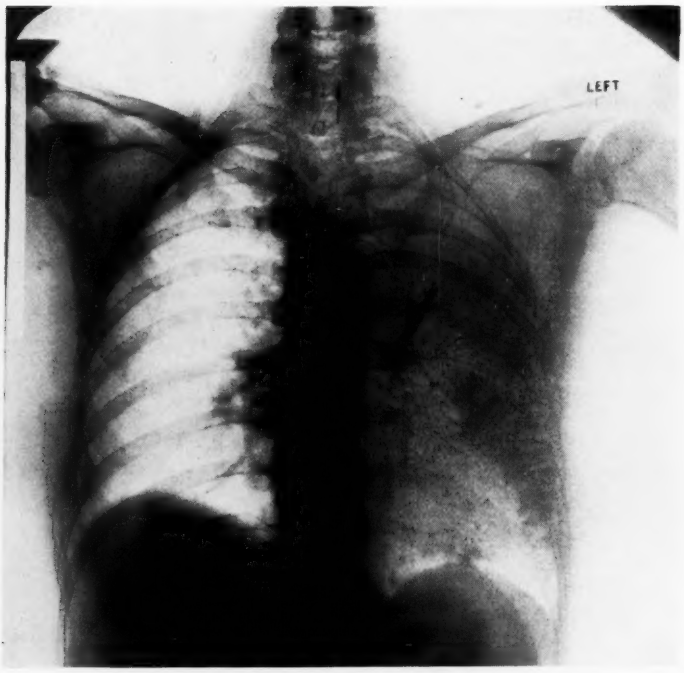


Fig. 4. Case 3. January 2, 1932. Postero-anterior stereoscopic films of chest. Lateral view of chest, left side to film.

There is almost complete opacity over the upper half of the left lung field from the anterior end of the first rib to the anterior end of the fourth rib. From this point downward along the left cardiac border to the diaphragm there is great haziness. The left cardiac border is lost in the opacity above the anterior end of the third rib. The cardiac outline does not appear to be enlarged. There is some increase in hilus on the right and generalized widening of lung markings which have in association a moderate amount of atypical coarse, deep, parenchymatous mottling. There is some calcification in the right hilus region. The right leaf of the diaphragm is rounded and the costophrenic angle is clear. In the lateral view, left side to film, the opacity seems to involve only the left upper lobe, forming roughly a triangle extending from a point posteriorly at about the fourth dorsal vertebra obliquely across the chest anteriorly to the dome of the diaphragm. The left lower lobe in the lateral view has good air content.

X-ray diagnosis: Carcinoma of lung.

The patient again entered the hospital in May, 1933, with complaints similar to those on previous admissions except that she had had increasing hemoptysis. She was bronchoscoped twice more, but a positive diagnosis of carcinoma was not obtained because of persistent and severe bleeding. It was felt unwise to try to get more tissue bronchoscopically or to try to deal with the tumor by this method, even though it did move within the bronchial lumen. External surgery was, therefore, recommended.

On January 3, 1933, the pleural cavity was opened by Dr. Evarts Graham and the mass in the right lower lobe explored. The pleura over the upper lobe was

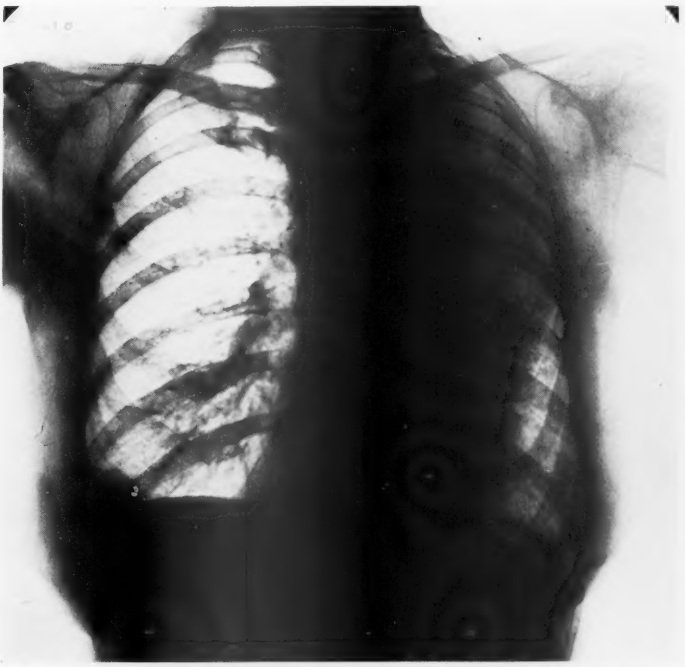


Fig. 5. Case 3. March 9, 1932. Anteroposterior view of chest, supine.
Anteroposterior view of chest, patient lying on left side.
Lateral view of chest.
Films taken for bronchography.
See films and reports, dated January 2, 1932.

There has been a marked increase in size of opacity noted in the left lung field, so that at this time the lung field is almost completely opaque, except for a small area just above the left leaf of the diaphragm, well out toward the lateral chest wall. The lower portion of the trachea and left main bronchus and some of its major divisions are fairly well outlined with opaque material. There is a rather large area over the seventh and eighth posterior ribs on the left, well out toward the lateral chest wall that shows an alveolar distribution of the oil.

X-ray diagnosis: As before.

rubbed vigorously with a gauze sponge in order to stimulate formation of adhesions. Approximately ten days later the chest was reopened and the right lower lobe was removed. At this time very free bleeding occurred when the tumor mass was manipulated. Apparently there was a weak spot in the wall of one of the larger bronchial vessels which under ordinary conditions was blocked by the pressure from the tumor mass. The patient died from shock.

Autopsy revealed carcinoma of the right lower lobe, atelectasis and fibrosis of the upper lung lobes on the right side.

CASE 3. S. R., white, female, aged 64, housewife, was admitted to Barnes Hospital January 3, 1934.

She had been perfectly well previous to February, 1931, when she began to have a dry, hacking cough from which she was unable to obtain relief. At about

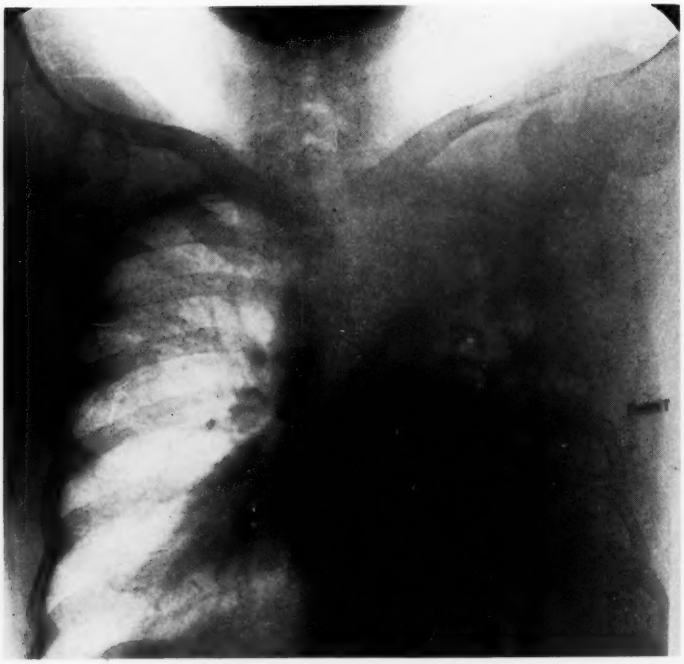


Fig. 6. Case 4. September 8, 1933. Anteroposterior view of chest, supine. See previous films and report.

There is almost complete opacity over the entire right lung field, except for multiple rounded dark areas of rarefaction in the second, third, fourth and fifth anterior interspaces. There is displacement of the heart and trachea to the right and narrowing of all the intercostal spaces on this side. The right leaf of the disphragm is obscured by the opacity of the lung field in the lower portion of the chest. Except for generalized thickening of all lung markings throughout the left side the parenchyma is relatively clear. There is an irregularity of the left leaf of the diaphragm but the costophrenic angle is clear.

X-ray diagnosis: Atelectasis, right lung. Multiple lung abscesses. Cardiac and tracheal dislocation.

the same time she noticed that she was easily fatigued. There was no weight loss and at no time had the cough been productive. There was never blood in the sputum. The past family history was essentially negative.

Physical examination revealed a rather obese white female, not acutely ill. The general physical examination was essentially negative except for the chest where there was dullness and many râles with diminished breath sounds in the left upper chest anteriorly and posteriorly.

Laboratory findings were essentially negative with the exception of 4-plus sugar in the urine.

X-ray, reported in legend accompanying plate, revealed a hazy mass in the left upper chest and lipiodol introduced at this time showed a blocking in the left upper lobe bronchus.

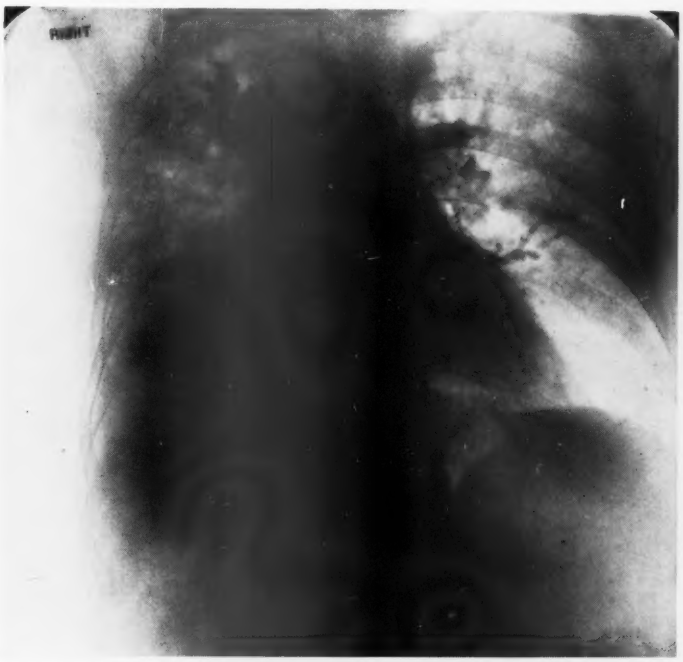


Fig. 7. Case 4. September 13, 1933. Anteroposterior view of chest—bronchography.

See previous films and reports.

A moderate amount of opaque material is seen in the lower portion of the trachea, just above the carina, and in the regions of the right and left main bronchus. There is extensive puddling of the opaque material in the dilated bronchi in the upper third of the right lung field. There is incomplete filling of some of the descending branches in the mid-portion of the left lung field. No evidence of opaque material in the descending branches on the right, though multiple irregular dark areas previously described are again visualized and have the appearance of cavities.

X-ray diagnosis: As before. Bronchiectasis, right.

G-I series was negative. The patient was bronchoscoped and the left upper lobe bronchus was found to be compressed, but no tumor tissue could be seen.

The patient was discharged from the hospital and died on the 9th of August, 1932.

No autopsy was permitted.

CASE 4. J. Y. B., white, male, aged 54, coal dealer, entered Barnes Hospital September 4, 1933.

Three years previously he had begun to have afternoon and evening elevation of temperature with occasional night sweats. He gradually became rather weak and noticed some shortness of breath on exertion. The patient had had chronic cough for some twenty years. This, however, had become worse and he had begun occasionally to raise small amounts of blood. In the morning he had raised a large

amount of tenacious, frothy sputum, which gradually began to acquire a foul odor. Patient had lost thirty-four pounds. Hemoptysis had become more frequent during the six months previous to admission.

Physical examination revealed a poorly nourished white male. There was dullness over the right chest, both anteriorly and posteriorly. There were many râles and rhonchi over right lung, with increased whispered voice sounds over same area. The left chest was normal.

Laboratory findings were essentially negative.

Neurologic examination revealed atrophy of the optic nerve on the right with positive Oppenheim and Babinski on the right. It was felt that the patient had either metastatic tumor or abscess in his temporoparietal region.

X-ray findings reported in legend accompanying plates.

Physical diagnosis was atelectasis of the right lung with multiple lung abscesses.

The patient was bronchoscoped and cancer tissue was found filling the right main bronchus and encroaching on the lumen of the left main bronchus. The lumen of the right bronchus was almost completely obliterated. Biopsy taken at the time from the bronchus revealed bronchogenic carcinoma. Lipiodol introduced reported with plate.

Patient was discharged from the hospital on September 16th and died at his home approximately six months later.

CONCLUSIONS.

In summarizing the points of special interest in the diagnosis of cancer of the lung, we have the following:

A. History, which has been discussed.

1. The peculiar type of cough.

B. Atelectasis C. Bronchiectasis D. Tuberculosis, not proven	Occurring clinically or apparently in middle life; otherwise unexplained.

The above mentioned changes may be demonstrated and localized by physical and radiologic study, but the true nature of the lesion can be proven in the early stage only by bronchoscopic study and biopsy. Endobronchial cancer may remain localized for a long period. The course and duration of parenchymal tumors is much more rapid than is that of hilar tumors.

This procedure is of value not only as a means of obtaining diagnostic information but in addition is often a means of rendering important therapeutic aid. The necessity for repeated examination in some cases is emphasized.

It is true that such studies are accompanied by a certain amount of risk, but this is negligible with proper safeguards. The results com-

pare favorably with exploratory methods of study of disease in other inaccessible regions of the body. With the improved methods in thoracic surgery and the resultant possibility for dealing with lung cancer by surgical removal, the value of early and accurate diagnosis is obvious.

539 North Grand Blvd.

BIBLIOGRAPHY.

1. Graham, E. A., and Singer, J. J.: Successful Removal of an Entire Lung for Carcinoma of the Bronchus. J. A. M. A., 101:1371-74, Oct. 28, 1933.

2. Meakins and MacLeod: Carcinoma of the Bronchi. Can. Med. Assn. J., 28: 268-75, 1933.

3. Graham, Evarts A.: The Diagnosis and Treatment of Primary Carcinoma of the Bronchus or Lung. Amer. J. Roentgen and Radium Therap., XXXL, No. 2:145-52, February, 1934.

4. Graham, Evarts A.: Ibid.

5. Weller, Carl V.: The Pathology of Primary Carcinoma of the Lung. Arch. Path., 7:478-519, 1929.

6. Jackson, Chevalier: Peroral Endoscopy and Laryngeal Surgery. The Laryngoscope Company, 1915.

7. Singer, J. J.: A Simple Method of Introducing Iodized Oil into the Lungs. J. A. M. A., Oct. 16, 1926, Vol. 87, 1298-1299.

8. Singer, J. J.: Ibid.

9. Carlson, Herbert A., and Ballon, Harry C.: The Operability of Carcinoma of the Lung. J. Thor. Surgery, 2:323-48, April, 1933.

CIV.

RESECTION OF THE CERVICAL AND UPPER THORACIC ESOPHAGUS FOR CARCINOMA: REPORT OF A CASE.*

FIELDING O. LEWIS, M. D.,

PHILADELPHIA.

Perhaps this phase of esophageal cancer is somewhat out of place to present before this Society, but since all Bronchoscopists are fundamentally concerned with this fatal disease and on them depends the accurate diagnosis, I feel justified in discussing before you a surgical procedure which has in the past been successful and which promises, I believe, a more favorable outcome in the future.

Statistics show that in between 3 and 5 per cent of all carcinoma deaths, the lesion is found primary in the esophagus. All reports reveal that this disease is far more prevalent in the male than in the female. In a series of two hundred cases examined in the Radiological Department of the Philadelphia General Hospital twelve (6 per cent) were women. In more than 50 per cent of reported series, the disease was located at or just above the cardia. The cervical and upper thoracic portions are the least involved. In our series eight (4 per cent) were located in this region; all were men. Vinson,[1] reporting one thousand cases, found that in about 5 per cent of the men, the lesion was located at the introitus, whereas, in women, approximately 30 per cent were found in that location.

The difficulty experienced in making an early diagnosis of esophageal cancer has been no doubt the chief hindrance in the successful advancement of its treatment. All workers in this field have emphasized this fact. Some believe that it is always in an advanced stage before symptoms are noticed and therefore early recognition is impossible; that at this stage it has invaded neighboring structures or has metastasized to the lymph nodes of the posterior mediastinum or other organs. So they apparently inquire, why attempt any radical form of treatment? Why not consider it hopeless and rely upon palliative measures? I believe that we should adopt a more optimistic view.

Not so very long ago, practically all cases of extrinsic cancer of the larynx were considered hopeless, but today many of them are

*Read before the seventeenth annual meeting of the American Bronchoscopic Society, Cleveland, June 11, 1934.

saved by radical surgery, plus irradiation. It is true that the vast majority of patients suffering with cancer of the esophagus are not seen by the esophagoscopist or surgeon until their condition is desperate, but if the family physicians were cognizant of the first symptoms and realized the importance of a careful examination, I believe it would be possible to recognize the condition much more frequently at an early stage. I should like to emphasize the view expressed by Professor Grey Turner,[2] who says, "It should be remembered that the first symptoms may be an attack of difficulty in swallowing, which may pass off completely. This should be looked upon as a warning and an absolute indication for careful investigation." He also adds that, "All surgeons ought to be familiar with the occasional, yet outstanding, successful results of radical surgery." I should prefer to include not only the surgeon but the medical profession in general especially the esophagoscopists, radiologists and the general practitioners.

The reported autopsy findings on patients who have died of this disease vary considerably. By some, practically all have shown definite metastasis, while other reports have shown no metastasis in from 20 to 48 per cent. A recent autopsy of one of our patients revealed an extensive local esophageal lesion without gross evidence of metastasis. However, the future success in its treatment depends on its early recognition; so it is hoped that in the near future some instrument of precision may be developed or some test discovered that will supply this much needed information.

Esophageal cancer is still a fertile field for experimental treatment. For many years the thoracic surgeons have by painstaking efforts and research attempted to establish a suitable and reasonably safe approach for its complete excision. The esophagoscopists in cooperation with the radiologists, have developed a meticulous technic for the application of radium in varying degrees of dosages. Roentgenologists are striving by means of the high voltage apparatus to pour sufficient x-ray energy into the growth for its complete destruction and restoration of function, with the result that up to the present so few have survived or been cured that those who have survived or been cured may be considered, perhaps, as curiosities. Consequently, there are many who recommend and practice only palliative measures such as intubation, dilatation or gastrostomy. It is not my purpose to discuss the relative value of these different forms of treatment. However, from the literature it would seem that surgical treatment has, up to the present time, given the most encouraging results. Especially is this true when the disease is located in the cervical portion.

One cannot read without being fascinated Torek's[3] report of the woman whose esophagus was removed by the trans-thoracic approach and who lived comfortably for more than thirteen years with the aid of an external rubber esophagus and died from pneumonia at a ripe old age. One's imagination cannot help being aroused by the recent report of Professor Grey Turner,[2] in which he describes the enucleation of the whole esophagus and the construction of a new esophagus by making a tube from the skin over the front of the chest which functions satisfactorily. These reports are alluring to the thoracic surgeon, but fail, perhaps, to inspire the laryngeal surgeon, but the laryngeal surgeon interested in cancer is encouraged by the report of a twenty-three year cure of cancer after the removal of the pharynx and cervical esophagus, as in the case of Arthur Evans, and the successes that are reported by A. Logan Turner of cancer in the same region. This means working in familiar territory and rightly comes, I consider, within the field of the laryngologist.

Of the two hundred cases of esophageal cancer in which I have been interested the vast majority, when first seen, were in a hopeless condition, emaciated and dehydrated. As aforementioned, in only eight cases in our series was the lesion found located in the cervical portion of the esophagus and of these only one was considered suitable for excision.

REPORT OF CASE.

CASE 1.—J. D., a white man, 43 years of age, a brewer by occupation, was first seen in the laryngeal clinic of the Philadelphia General Hospital, in October, 1933. His chief complaint was difficulty in swallowing. He had been well until about three months previous to his first visit, when he began to have difficulty in swallowing solid foods. He modified his diet, but the difficulty increased until even soft foods could not be taken. For the past three weeks he had been able to take only liquids. The rest of his history was negative, except that his father died of carcinoma of the esophagus. His physical examination and laboratory reports were quite satisfactory. X-ray report is as follows: "There was no obstruction to the standard barium meal. Barium filled capsule met with obstruction at the level of the first dorsal vertebra. This could not be washed down with water. X-ray conclusions: Stricture of the esophagus at the level of the first dorsal vertebra, probably malignant."

"Fluoroscopic examination showed no hesitation nor difficulty in swallowing barium. No fixation of larynx and during the swallowing act the larynx rises and there is no spill-over at any time in the laryngeal vestibule. In the antero-posterior view, there is no evidence of the adherence of any of the barium in either pyriform sinus or at any point along the esophagus. Barium passed readily into the stomach. There may be a slight degree of retrotracheal swelling."

The bronchoscopic clinic reported: "There is an obstruction at the level of the cricopharyngeus. There is a proliferation of tissue which bleeds easily on touch, and which almost entirely blocks the lumen of the esophagus." Biopsy

taken at this time, was reported as squamous cell carcinoma, type three, of the esophagus. He was then referred to the radiologic department for treatment. November 15th, 1933, a gastrostomy was performed under local anesthesia, following which the patient's general condition greatly improved.

Taking into consideration the patient's age and excellent physical condition, with no perceptible glandular metastasis, it was decided to make an attempt at resection of the diseased portion of the esophagus. Before doing so, however, I was given ample opportunity and assistance, through the courtesy of Dr. O. V. Batson and Dr. J. Parsons Schaeffer, to study the approaches as described by others on several cadavers and was greatly impressed by the approach demonstrated to me by Dr. O. V. Batson, which is as follows:

"In the lateral cervical approach to the upper esophagus advantage is taken of the surface of cleavage between the vertebral column with its muscles and the neck viscera. This surface of cleavage is a collection of loose areolar tissue lying between the very mobile esophagus and the fairly rigid cervical structures. This cleavage extends above, to the base of the skull, and below, far down into the posterior mediastinum. Laterally, in the neck region, it is covered by little except the skin, platysma and capsular neck fascias.

"For approach to the esophagus a skin incision is made at the posterior border of the sterno-cleido-mastoid muscle from the clavicle upward, two-thirds the length of the muscle. Right or left side may be used. The nearness of the esophagus to the left side is more technical than real, and any fancied advantage that this nearness might give is overbalanced by the relations of the thoracic duct to the esophagus on the right side and the possible trauma to the left vagus. Unless there are some particular reasons in a given case the right side is preferable.

"The incision is carried through the platysma. Here are encountered the external jugular vein and the cutaneous nerves of the cervical plexus. To give adequate exposure, the external jugular vein is cut between ligatures. The cutaneous colli nerve, though probably not visualized, is severed at this time along with some of the suprasternal cutaneous nerves.

"The sterno-mastoid muscle is retracted anteriorly, and with a slight flexion of the neck, the entire visceral portion, including the jugular vein, carotid artery and accompanying nerves, go forward with the muscle.

"By carrying a dissecting scissors or a Kelly forceps into the incision, staying in contact with the posterior structures (the sca-

lenus anterior and the longus colli muscles and their overlying prevertebral fascia), the sagittal partition of Jonnesco is encountered and penetrated. This opening admits the instrument into the areolar area of cleavage. With the finger in the space the vertebral column with its muscles, the scalenus muscles, the brachial plexus and the vertebral vessels lie below. Above the finger lie, among others, the carotid arteries, the jugular veins, the sympathetic chains, the vagi, the esophagus, trachea and sterno-mastoid muscles.

"The opening in the sagittal partition is enlarged to permit adequate retraction and vision. When this is accomplished, the esophagus is freed from the anterior visceral mass by blunt dissection. It may be clamped and severed so that delivery of the lower portion is expedited. As long as the procedures are confined to the neck, the only special precaution that should be mentioned is the possible damage to the neck vessels by a too vigorous or too prolonged retraction.

"When the dissection is carried into the mediastinum it must be remembered to work toward the midline rather than laterally, for the vertebral vessels, the subclavian vessels and the sympathetic chains go from and anterior-lateral to a posterior-lateral relationship. This they do at a line with the lower end of the sagittal partitions.

"With but these two precautions, of an anatomic character, it is possible to follow the esophagus and free as far down as the level of the angle of the sternum (angle of Ludwig)."

Consequently, on March 3rd, 1934, under small dose of avertin anesthesia, supplemented by intratracheal gas ether, the above described approach was made. The esophagus was easily dissected posteriorly from the prevertebral fascia. Anteriorly, it was found that the disease had penetrated the wall of the esophagus and involved the posterior wall of the trachea from which it was with difficulty dissected. The lesion extended from the introitus to below the second dorsal vertebra. A clamp was placed above the lesion at the entrance to the esophagus as well as below the area which was thought to be distal extent of the disease. The portion of the esophagus between the two clamps was then removed. The clamps were left in position for seventy-two hours. A capsule of one-half millimeter silver and one millimeter brass, containing 183 mg. of radium was placed in contact with the involved portion of the posterior wall of the trachea, a total of 2745 mc. hours was given. Cigarette drains were placed in the posterior mediastinum and the wound packed with iodoform gauze.

The patient was making quite a satisfactory convalescence, was out of bed and around the ward during the second week. Four weeks and five days after the operation, the patient died from a severe and uncontrolable hemorrhage. Postmortem findings were very interesting and instructive. There was no evidence of infection in the posterior mediastinum, which had been well walled off at the point where the esophagus had been severed. The distal end of the esophagus, however, was only partially closed. The pharyngeal end was likewise partially

open, which had been evident before death from the constant drainage of siliva from the wound. The application of radium to the involved portion of the trachea had evidently produced the desired result, as no remains of the growth was apparent. There was an acute suppurative arteritis with necrosis and perforation of the right subclavian artery, which caused death. Histologic examination of the esophagus revealed incomplete resection of the carcinoma, as the distal segment showed invasion with squamous cell carcinoma by direct extension and lymphatic permeation.

In spite of the fatality experienced in this case, I am still optimistic as to the possibility of the surgical removal of carcinoma of the cervical and upper thoracic esophagus by this approach. In fact, I believe resection as far down as the tracheal bifurcation is feasible. My failure to sever the esophagus below the involved area could, I think, have been avoided had I been equipped with electrically lighted retractors. Should a similar case be encountered, I should also attempt to close the distal end of the esophagus by invagination and the introduction of a purse string suture.

259 SOUTH 17TH ST.

REFERENCES.

1. Vinson, Porter P.: "Malignant Disease of the Esophagus." Northwest Medicine, 8:320-323 (August), 1933.

2. Turner, Prof. Grey: "Recent Advances in the Treatment of Carcinoma of the Esophagus from the Surgical Aspect." The J. of Laryng. and Otol., 49:297-312 (May), 1934.

3. Torek, Franz: "Surgical Treatment of Carcinoma of the Esophagus." Arch. Surg., 12:232-235, Part 2 (January), 1926.

CV.

CARCINOMA OF THE ESOPHAGUS, ITS DIAGNOSIS AND TREATMENT.*

WILLIAM A. HUDSON, M. D.,

DETROIT.

Recent years have seen the breaking down of many barriers to the successful treatment of diseases that formerly were considered to be outside the realm of medical aid. Only a few years ago we began to discuss with earnestness and with a degree of hope the operation of pneumonectomy for carcinoma of the lung. Today we are continually hearing reports of new series of cases that have been subjected to surgical removal of a part or all of a lung for carcinoma. Already there is a group of cases that have lived long enough following operation to warrant the thoracic surgeon in facing the future with a high degree of hope. Is it any surprise then that one should now turn one's attention to the study of a lesion that has been looked upon in the past as having a mortality of practically 100 per cent regardless of the type of treatment employed.

Carcinoma of the esophagus is undoubtedly one of the most benign of the carcinomata with which we have to deal, yet in the minds of most men it remains today the same hopeless disease as of its earliest days, and until some new method of treatment of cancer is discovered we must look to the surgeon for hope for cure.

It seems to us that the fundamental reasons for the shocking death rate from carcinoma of the esophagus lie in the fact:

1. That we have in the past been so busy trying to overcome other problems that seemed to be difficult that we have not devoted the necessary time to the study of the problems of carcinoma of the esophagus.

2. We have been afraid to know the truth about esophageal lesions—that is, we knew that some esophageal lesions were nonmalignant and we preferred to consider all lesions nonmalignant until they had proven themselves to be malignant. The safer way would have been for us to consider all unexplained esophageal disturbances

*Read before the seventeenth annual meeting of the American Bronchoscopic Society, Cleveland, June 11, 1934.

as having been of carcinomatous origin until an otherwise satisfactory explanation had been offered.

When we pull our heads out of the sand and face the situation squarely and study even the earliest symptoms from the standpoint of their being caused by a malignant disease, we will find that instead of the patient giving us a history of marked dysphagia, pain, cachexia and emaciation, he will describe a very slight abnormal sensation in the cervical, retrosternal or epigastric region. We will find not the far advanced, fungating, ulcerative and stenosing lesions but the very early lesions which have not produced obstruction and they will be free of ulceration. The patient will be in good general condition.

We would urge the early recourse to diagnostic roentgen ray measures, but with a negative x-ray study in the presence of definite symptoms that are not fully and satisfactorily explained one must resort to actual inspection of the inside of the esophagus. In this examination particular attention should be given to those sites at which the lesion most frequently occurs.

In those cases in which the esophagoscopic examination fails to show the presence of a definite lesion, the patient must be kept under observation and the examination must be repeated after a short interval, especially if the symptoms persist. In those cases in which a lesion is found biopsy should be taken to determine the character of the lesion.

Operations upon the cervical esophagus have resulted in an operative mortality of from at least 18 to 22 per cent, according to Graham,[1] while of all the cases operated upon few have survived for any length of time. However, it seems to us that when we consider the fact that Gluck had six patients living five years after operation out of 114 operated upon for carcinoma of the upper third of the esophagus, we should see a ray of hope for the future, especially if we can only see these cases earlier. There have been similar cases of carcinoma of the lower third of the esophagus operated upon with a degree of success. Five cases were found by Graham which were operated upon through the abdominal route by Bircher, Kummell, Kuttner, Volcher and Brun, while Zaaijer and Hedblom each operated upon a case; the former's died of recurrence while the latter's committed suicide. These two portions of the esophagus have been considered the most accessible portions. The thoracic or middle third has been considered the most baffling because of the difficulty involved in repairing the defect that remains after the lesion has been removed. We all know of the remarkable case operated upon by Doctor Toreck,[2] whose patient lived for thirteen years following the

operation, and of the case operated upon by Doctor Lilienthal,[3] who lived for one year and four months before dying of recurrence, and of Egger's[4] successful operative recovery. Alexander[5] also has a case that successfully recovered from operation. Certainly with the encouragement offered through this degree of success it seems to us that we should, in a manner of speaking, forget about the repair of the esophageal defect for the time being and consider only the measures that will enable us to eradicate the lesion and preserve life with a degree of comfort to the patient. Once we have definitely established the patient on his feet free of carcinoma we have the rest of his life to worry about reconstructing a tube for feeding purposes.

Our experience in resection of the esophagus for carcinoma have involved four patients. They had all had careful x-ray and esophagoscopic studies, including tissue studies, to confirm the diagnosis.

REPORT OF CASES.

CASE 1.—A white female, 64 years of age. Her lesion was in the middle third of the esophagus just about one inch below the arch of the aorta. A gastrostomy was performed for feeding purposes, then nine days later, by the left transthoracic route, the esophagus was exposed, the mediastinal pleura being incised just anterior to the descending aorta. By blunt dissection the esophagus was freed throughout its middle third, the dissection being carried behind the arch of the aorta. Then a second incision was made in the mediastinal pleura above the arch of the aorta and that portion of the upper third of the esophagus which lay within the mediastinum was freed. Double ligatures were placed about the esophagus at the junction of the middle and lower third of the esophagus and the tube sectioned between the ligatures. The distal stump was treated with carbolic and alcohol and inverted much the same as an appendix stump. The middle third with the lesion, which was about one and one-half inches in its greatest length and did not extend outside the wall of the esophagus, was dragged upward behind the arch of the aorta and left dangling through the second mediastinal incision free with its ligature in the pleural space. It was not necessary to use any ligatures for hemostasis. The mediastinal pleura was brought together over the distal stump and the opening through the chest wall was closed. An incision about one to two inches long was made at the lower medial insertion of the left sternocleidomastoid muscle and by blunt dissection the left pleural space was entered. The ligature was grasped and the freed esophagus was brought out through the wound in the neck. Closure of this wound was in layers with the carcinomatous portion of the esophagus and a good margin of the healthy tissue being cut away. The patient left the operating room in satisfactory condition. She died twenty-four hours later. At postmortem it was discovered that the gastric juices had digested much of the fat in the left abdominal wall. The operative wounds from the esophagectomy were clean and the stump of the distal end of the esophagus was sealed. There was no free fluid in the left pleural space. No evidence of metastasis, lymph gland, or otherwise was encountered.

CASE 2.—A white male, near 65 years of age, was explored by the transthoracic route in hopes that a resection might be carried out for a carcinoma of

the middle third, but the lesion was found to extend beyond the wall of the esophagus to involve the mediastinal pleura on the left. The wounds were closed. The patient died a few weeks later from inanition.

CASE 3.—A white male, 68 years of age, whose lesion involved the lower portion of the middle third of the esophagus, was operated upon by the left transthoracic route and the middle and lower third of the esophagus were resected in much the same manner described in Case 1. In this case, however, the dissection was somewhat more difficult than usual for that portion of the esophagus which lay behind the heart. The patient left the operating room in poor condition and died within three hours from surgical shock. However, the lesion had been cleanly removed.

CASE 4.—A white male, 41 years of age, was operated under novocain anesthesia for a carcinoma of the upper third of the esophagus. The lesion involved about 1½ inches of the esophagus at and below the cricopharyngeal constrictor, but did not involve the larynx. In this case the incision was made along the medial margin of the left sternocleidomastoid muscle, and the esophagus was identified and freed below the lower level of the lesion. The lesion was freed by blunt and sharp dissection. Bleeding points were ligated. When the dissection was completed the upper limits of the dissection extended to a point just above the cricopharyngeal constrictor. The lower limit was at a point just above the upper margin of the sternum. This entire section of the esophagus between these points of limitation was removed. The upper end of the distal stump was attached to the skin and the lower portion of the wound was closed while the upper half of the wound was packed firmly with dry gauze. There was no respiratory disturbance. The patient left the operating room in very good condition but died suddenly two hours later with evidence of cerebral embolus. Postmortem examination revealed no evidence of metastasis.

In retrospect, one might be much discouraged to find that all the patients have died, but it seems to us that the causes of death have been such that with earlier diagnosis and with certain minor changes in the operative technic we can face the future with hope.

Our experiences have led us to the decision that any patient with a carcinoma of the esophagus which seems to be limited to the esophagus is entitled to a surgical exploration and, if at all possible, resection of the lesion.

819 DAVID WHITNEY BUILDING.

REFERENCES.

1. Graham, Evarts A., Ballon, Harry C.: Symposium on Cancer of the Esophagus. ANNALS OF OTOLOGY, RHINOLOGY AND LARYNGOLOGY, 40:895 (Sept.), 1931.

2. Toreck, F.: Surgery of the Esophagus. Nelson's Loose Leaf Living Surgery, 1927, Vol. 4, p. 119.

3. Lilienthal, H.: Carcinoma of the Thoracic Esophagus, Extra Pleural Resection and Plastic. Ann. of Surg., 74:259, 1921.

4. Eggers, C.: Resection of the Thoracic Portion of the Esophagus for Carcinoma. Arch. of Surg., 10:361, 1925.

5. Alexander, John: Personal communication.

CVI.

OBSTRUCTIVE ATELECTASIS FOLLOWING REMOVAL OF FOREIGN BODY FROM THE LUNG DUE TO VISCID SECRETION AND THE INHIBITION OF COUGH BY MORPHIN.*

GABRIEL TUCKER, M. D.,

AND

CHARLES C. WOLFERTH, M. D.,

PHILADELPHIA.

The question as to just what part obstruction to the bronchus plays in obstructive atelectasis of the lung, particularly in the type of atelectasis found in postoperative pulmonary complications, has been a matter of much conjecture and debate. The finding of obstructing secretion in the first case of massive collapse bronchoscoped and the observations from the study of the first case made by one of us and Dr. Walter Estelle Lee are well known. The experimental work done by Lee, Tucker, Clerf and Ravdin on this particular phase of obstructive atelectasis confirmed the observations and conclusions made.

In this case which we are reporting, the obstruction occurred without any of the factors that have been so much emphasized in discussions of the etiology and the mechanism of atelectasis occurring as a postoperative complication. The only factors present were outpouring of viscid secretion from the affected area of lung that had been previously blocked, and the use of morphin which inhibited the normal cough mechanism. Massive collapse occurred and was relieved by bronchoscopic removal of the secretion. The cough mechanism became effective when the morphin was withheld, and there was no further recurrence of atelectasis.

REPORT OF A CASE.

CASE 1.—A boy, 13 years of age, was referred to one of us and sent into the hospital of the University of Pennsylvania because of cough with hemoptysis. The cough was chronic and productive until shortly before admission when

*Read before the seventeenth annual meeting of the American Bronchoscopic Society, Cleveland, June 11, 1934.

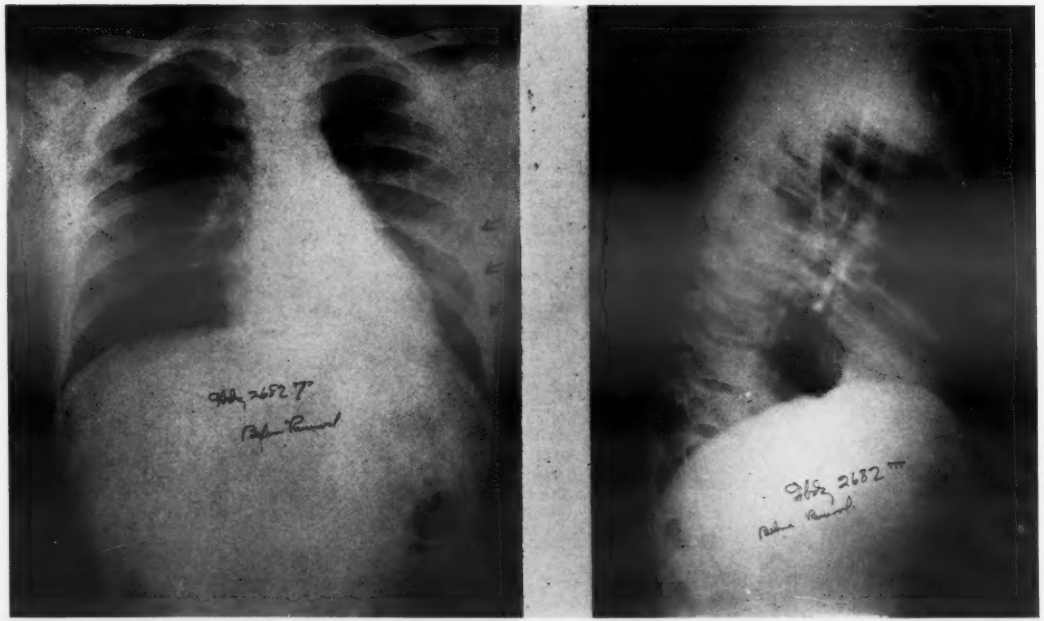


Fig. 1. Anteroposterior and lateral films showing density in the posterior portion of the left lower lobe. This was found to be due to atelectasis produced by the timothy head blocking a branch bronchus.

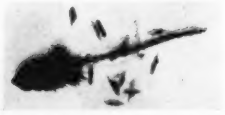


Fig. 2. Timothy head removed through the bronchoscope from the external division of the left lower lobe.

hemoptysis occurred. The history indicated a possibility that the patient had aspirated a timothy head about five years before, when, after a choking attack, the child, then about eight years of age, developed pulmonary signs that were thought to be due to a cold. The matter of the choking had been given very little attention and the child had gone along with a chronic cough and several attacks of "bronchopneumonia" involving the left lung. Finally, when the child began to spit up blood, the symptoms were given serious consideration. The child was then referred and clinical evidence of obstruction at the base of the left lower lobe found. This was confirmed by x-ray examination (Fig. 1) and a diagnostic bronchoscopic study was made.

On bronchoscopic examination the stem of a timothy head was seen in the lumen of an external terminal division of the left lower lobe bronchus. This branch bronchus was very much inflamed and filled with granulation tissue; blood stained secretion was seen to be coming from the area of lung drained by this branch bronchus. The timothy head (Fig. 2) was removed through the bronchoscope and considerable viscid secretion was aspirated from the left lower lobe. There was no reaction following the bronchoscopic examination and we were certain that no trauma had been inflicted. The patient was given morphin to quiet his cough by the interne, on his own responsibility. Much to our surprise, within the twenty-four hours following bronchoscopic removal of the foreign body, the patient developed a complete atelectasis of the left lung. X-ray examination by Dr. Karl Kornblum confirmed the physical findings (Fig. 3). Bronchoscopy was done without anesthesia and the left main and lower lobe bronchus were found to be filled with the same type of secretion that had been aspirated at the time of the removal of the foreign body. Following the bronchoscopic aspiration of this secretion the patient continued to cough up considerable secretion. All morphin was withheld and he was placed on his right side so that the proper postural drainage could be obtained. The atelectasis cleared very promptly and the lung expanded. Inflammatory reaction and granulations were noted in the bronchus where the foreign body had been located. The granulations were removed, and local treatment with aspiration of the left lower lobe area, which had been blocked by the foreign body, was carried out bronchoscopically. The atelectasis did not recur and the bronchial stenosis was cured by this local treatment (Fig. 4).

The evidence here that the atelectasis was due to the viscid secretion released from the diseased area of lung that had been blocked by the foreign body, plus the inhibition of cough due to the administration of morphin, is so clear and convincing that it was felt that the case was well worth reporting.

COMMENT.

1. A case of obstructive atelectasis due to secretion released from an obstructed area of lung by the removal of the foreign body, and inhibition of the cough mechanism by morphin, is reported.

2. Removal of the obstructing secretion by the bronchoscope followed by prompt clearing of the lung established beyond question

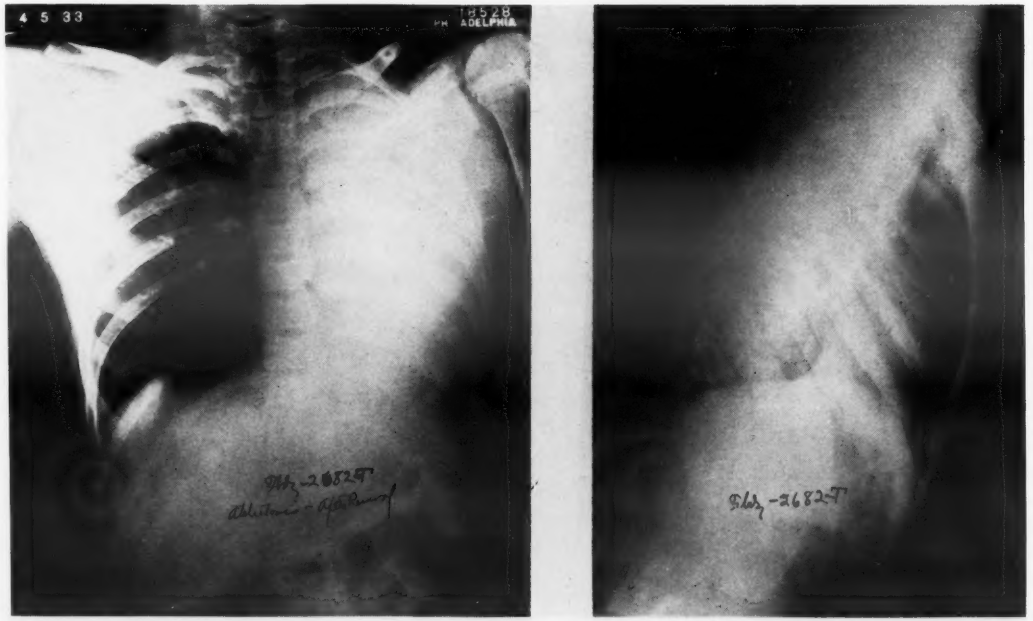


Fig. 3. Massive atelectasis of the left lung coming on twenty-four hours after removal of the foreign body. Note the increase in density of the left chest and the displacement of the heart toward the left beyond the midline.

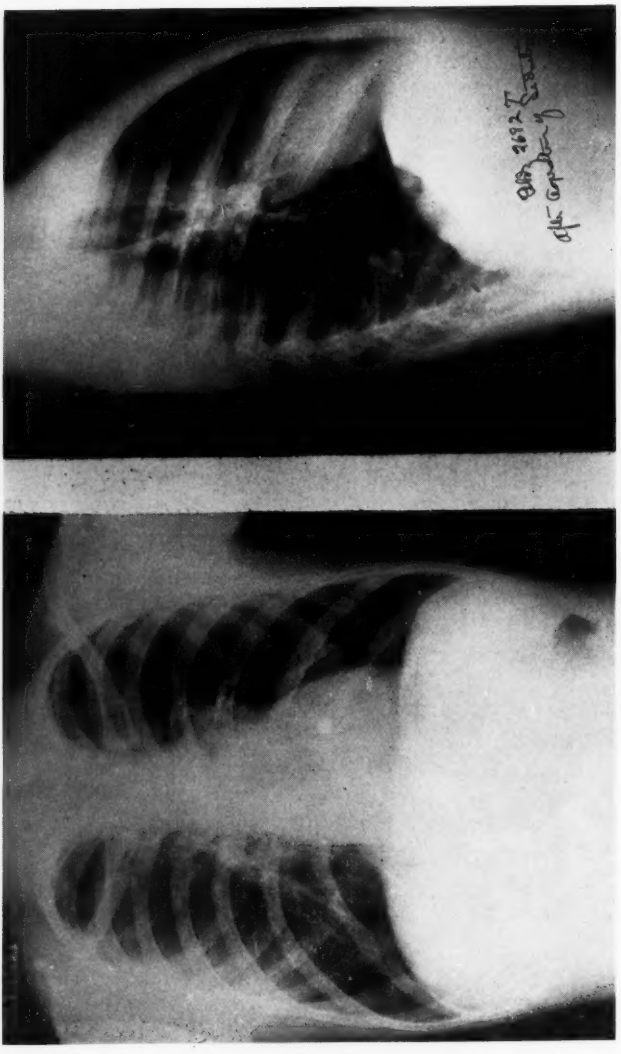


Fig. 4. Roentgenograms of the chest made following the bronchoscopic aspiration of secretion from the left lung. The left lung has completely expanded and the heart has assumed its normal position. There is no evidence of atelectasis. Atelectasis did not recur and the lung remained clear on both physical and x-ray examination. Roentgenograms made by Dr. Karl Kornblum.

that the atelectasis was due to the presence of obstructing secretion in the tracheobronchial tree. Withholding of morphin allowed the cough mechanism to resume its function and no further atelectasis occurred.

3. Inspection at the subsequent bronchoscopies that were given for treatment of the left lower lobe lesion, demonstrated that the condition was not due to bronchoscopic trauma.

326 South Nineteenth Street.

3600 Spruce Street.

CVII.

PRESENTATION OF SPECIMENS AND INSTRUMENTS.

AN EXTENSION BRONCHOSCOPE.

GABRIEL TUCKER, M. D.,

PHILADELPHIA.

The desirability of getting more deeply into the smaller branch bronchi, particularly in the lower lobes, has been felt by every bronchoscopist. A long, narrow tube being introduced through the mouth into the lower lobe bronchi has many disadvantages. A number of extension instruments have been devised, particularly of the proximally lighted types.

Fig. 1. Extension bronchoscope with one piece light carrier and lamp. Perforations in the tube permit breathing from the lung not under examination. The size tube illustrated is for 6 mm. by 40 cm. full lumen, and is 45 cm. in length.

The instrument I wish to present is to be used through a 6 mm. bronchoscope. We were able to construct this instrument with an adequate lumen because of the use of the one-piece light carrier and lamp which has previously been shown. The instrument is 45 cm. in length and is provided with a distal light, and has perforations for breathing far enough from the distal end to prevent obstruction to the breathing holes in the bronchoscope while it is in use.

---

*Read before the seventeenth annual meeting of the American Bronchoscopic Society, Cleveland, June 11, 1934.

CVIII.

AN EXTENSION ESOPHAGOSCOPE.

GABRIEL TUCKER, M. D.,

PHILADELPHIA.

The desirability of getting through a narrow lumen stenosis in the lower esophagus had been felt by everyone doing esophagoscopy. In order to accomplish this an extension esophagoscope has been devised carrying the one-piece light carrier and lamp. A tube 60 cm. in length has been constructed and can be used through a 9 mm. full-lumen by 45 cm. or a 9 mm. full-lumen by 60 cm. esophagoscope.

Fig. 1. Extension esophagoscopes 60 cm. in length are used through a 9 mm. full lumen esophagoscope. A one-piece light carrier and lamp gives ample lumen. The author's thumb valve is used on the suction canal.

(Fig. 1.) It is provided with a suction canal and the author's thumb valve to keep the field clear of secretion. Its use will permit the inspection of narrow strictured areas in the lower end of the esophagus. Smaller size extension esophagoscopes are also provided for 7 mm. and 6 mm. full-lumen tubes.

---

*Read before the seventeenth annual meeting of the American Bronchoscopic Society, Cleveland, June 11, 1934.

CIX.

A FIVE MM. FULL LUMEN THIRTY-FIVE CM. ESOPHAGOSCOPE AND GASTROSCOPE.

GABRIEL TUCKER, M. D.,

PHILADELPHIA.

In gastroscopy for foreign body in small children it is desirable to use the smallest size tube compatible with the work that is to be done through the tube because of the pressure exerted on the trachea during the procedure of gastroscopy. Because of this, an esophagoscope of 5 mm. full lumen diameter and 35 cm. in length was constructed, using the one-piece light carrier and lamp, as a companion instrument to the 4 mm. by 35 cm. infant gastroscope. This permitted the reduction of the outside of the tube, which reduction is

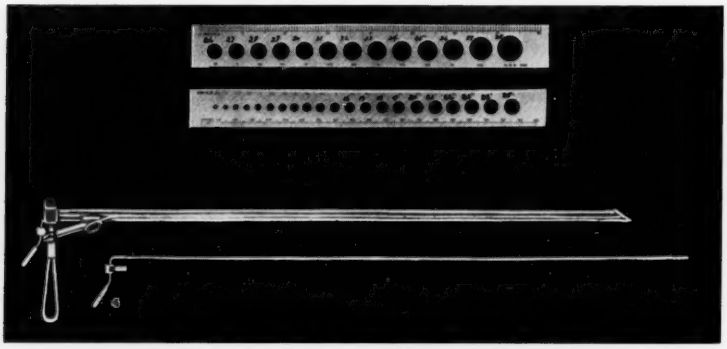


Fig. 1. A 5 mm. by 35 cm. full-lumen esophagoscope with one-piece light carrier and lamp, aspirating canal, with the author's thumb valve attachment. The outside size of this special tube is equal to 24 French scale. The regular 5 mm. full-lumen esophagoscope is equal in outside size to a 32 French scale. The tube is especially useful in esophagoscopy and gastroscopy in small children.

very desirable in preventing pressure on the trachea. The 5 mm. full lumen will permit the use of a curved forceps that will go around the corner in the stomach. The outside of this tube is equal in size to a 24 French scale. A regular esophagoscope, 5 mm. full lumen occupies a space equal to a 32 French scale. The tube is provided with the author's thumb valve attachment on the aspirating canal.

*Read before the seventeenth annual meeting of the American Bronchoscopic Society, Cleveland, June 11, 1934.

NOTICE.

The American Board of Otolaryngology.

An examination was held in Chicago, Illinois, September 8, 1934, during the meeting of the American Academy of Ophthalmology and Otolaryngology. Sixty-two candidates were examined, of which number, thirteen were conditioned or failed.

An examination was held in San Antonio, Texas, November 13, 1934, during the meeting of the Southern Medical Association. Twenty-five candidates were examined, of which number seven were conditioned or failed.

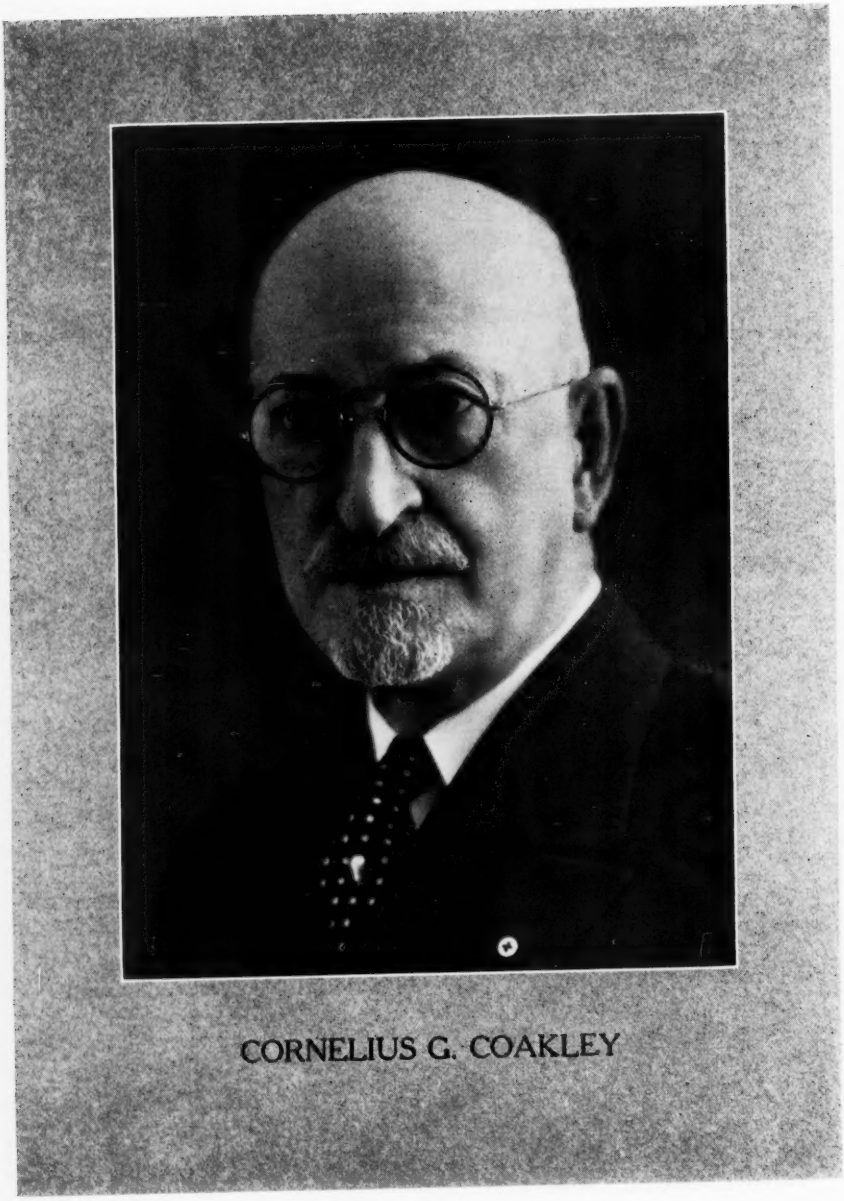
During 1935, examinations will be held in New York City, June 8th, during the meeting of the American Medical Association, and in Cincinnati, in the fall, at the time of the meeting of the American Academy of Ophthalmology and Otolaryngology. Prospective applicants for certificate should address the Secretary, Dr. W. P. Wherry, 1500 Medical Arts Building, Omaha, Nebraska, for application blanks.

W. P. Wherry, M. D., Secretary-Treasurer.

H. P. Mosher, M. D., President.

NOTICE.

The Medical School of Washington University, St. Louis, offers a week's intensive training in ophthalmology and otolaryngology. Only qualified ophthalmologists and otolaryngologists will be accepted for this course. The course begins on Monday, February 25th, and terminates on the evening of Saturday, March 2nd. For information, address the Registrar, School of Medicine, Washington University, St. Louis, Mo.



CORNELIUS G. COAKLEY

CORNELIUS GODFREY COAKLEY.

1862—1934.

Dr. Cornelius Godfrey Coakley was born in Brooklyn, August 14, 1862, eldest son of George W. Coakley, Professor of Mathematics and Astronomy at New York University, and Isabelle Hoe Coakley. He received the degree of Bachelor of Arts from the College of the City of New York in 1884 and of Master of Arts in 1887. The degree of Doctor of Medicine was conferred upon him by the New York University Medical College in 1887 when he graduated with first honors. He served as an interne in Bellevue Hospital and early began his distinguished teaching career as instructor in Histology and Anatomy in the school from which he graduated. Diseases of the ear, nose and throat proved especially interesting to him, partly because, at that time comparatively little was known of them. After study and research here and considerable work abroad, being chiefly impressed by the work of Professor Killian, he brought back the best of the knowledge acquirable in Europe. In 1896 he became Clinical Professor and in 1905 Professor of Laryngology at University and Bellevue Medical College, where he remained until 1914, when he accepted the chair of Otolaryngology at the College of Physicians and Surgeons of Columbia University.

His interest and ability in teaching and the clinical training of his students, internes and assistants never abated, and although inclined to be conservative, he kept abreast of any advances.

Associated with the late Dr. Caldwell, he made valuable experiments and some of the first useful radiographs of the nasal accessory sinuses.

Dr. Coakley's amazing energy and well directed effort enabled him to accomplish much hospital work. He organized and founded the Ear, Nose and Throat Service at Bellevue Hospital in 1916, where he continued as Director until he was asked to establish a similar service at Presbyterian Hospital on the opening of the Columbia Medical Center in 1928. Since then he has assiduously worked to improve the service to patients having ailments of the ear, nose and throat, establishing a ward in the Babies' Hospital a year ago. He was consultant to numerous other hospitals, including Bellevue, Woman's, Neurological Institute, Sloane Hospital, Southampton, Stuyvesant Square and Seaview hospitals.

Dr. Coakley, unfortunately, had no children. He was particularly fond of them and won the affection of his little patients by his engaging understanding of their viewpoints. He is survived by his widow, the former Miss Mary Louise Perry, niece of his first wife (nee Annette Perry), who died in 1922, in whose memory he established a scholarship at Vassar. Five sisters and four brothers also survive him.

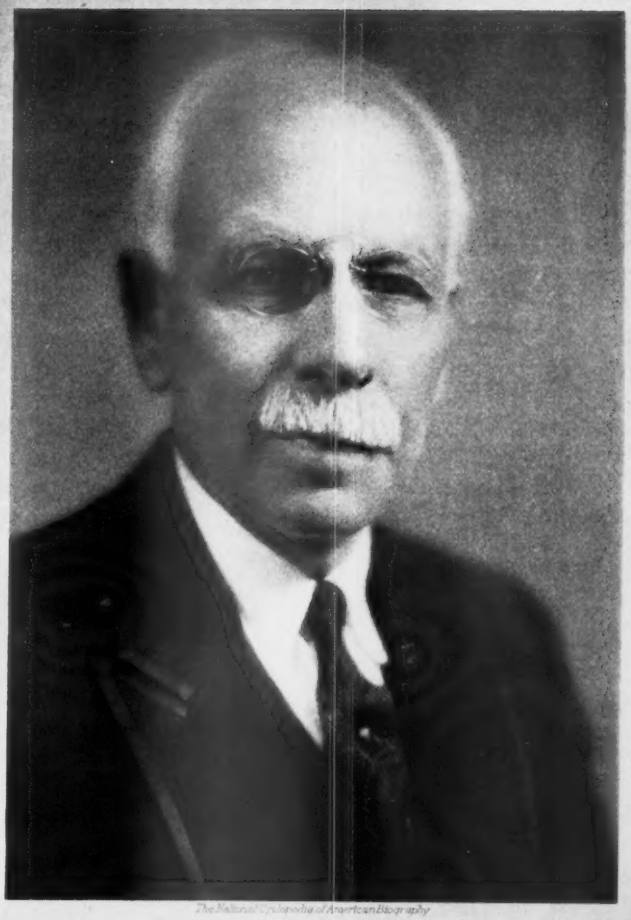
He was a hard worker and demanded as much from his subordinates, but when off duty was a most genial companion. He took great pleasure in all sports, particularly golf. He greatly enjoyed striving to beat one of his cronies on the links. This he usually could do, as only last summer he turned in a card of 78. He was a Mason, a member of Phi Gamma Delta, Phi Beta Kappa, the Mountain Lake Club of Lake Wales, Florida, and Lotus Club, the Campfire Club, the Long Island Country Club and the National Golf Links of America.

Among the professional societies he belonged to were the New York Academy of Medicine, since 1894; Medical Society of the County of New York, New York State Medical Society, American Medical Association, American Laryngological Society, American Otological Society, American College of Surgeons, American Laryngological, Rhinological and Otological Society, the Societé de Laryngologie des Hopitaux de Paris, of which he was elected a member after a series of lectures in Paris in 1924.

He was author of many articles in the field of his endeavor and a textbook used widely in medical schools, "A Manual of Diseases of the Nose and Throat," the seventh revision of which was published in 1930.

Dr. Coakley's ability as a teacher is best exemplified by the outstanding careers of the many men trained by him, now scattered throughout the country.

His brilliance in his special field has been widely appreciated, not only in this country, but in Europe and Japan. However, the memory that his host of patients, associates and friends chiefly hold of him is of his kindness, his helpful understanding of their difficulties and his unswerving integrity. JAMES W. BABCOCK.



The National Cyclopedia of American Biography

Wendell Phillips

WENDELL CHRISTOPHER PHILLIPS.

1857-1934.

Dr. Wendell Christopher Phillips, former president of the American Medical Association, died in Manhattan Eye, Ear and Throat Hospital after a short illness. Dr. Phillips, who was 77 years old, had practiced medicine in New York City for fifty-two years and, in addition to his wide experience as a physician and an officer of many medical societies, was foremost among the leaders of social work for the hard of hearing in securing recognition of the health and educational problem of the hard of hearing child.

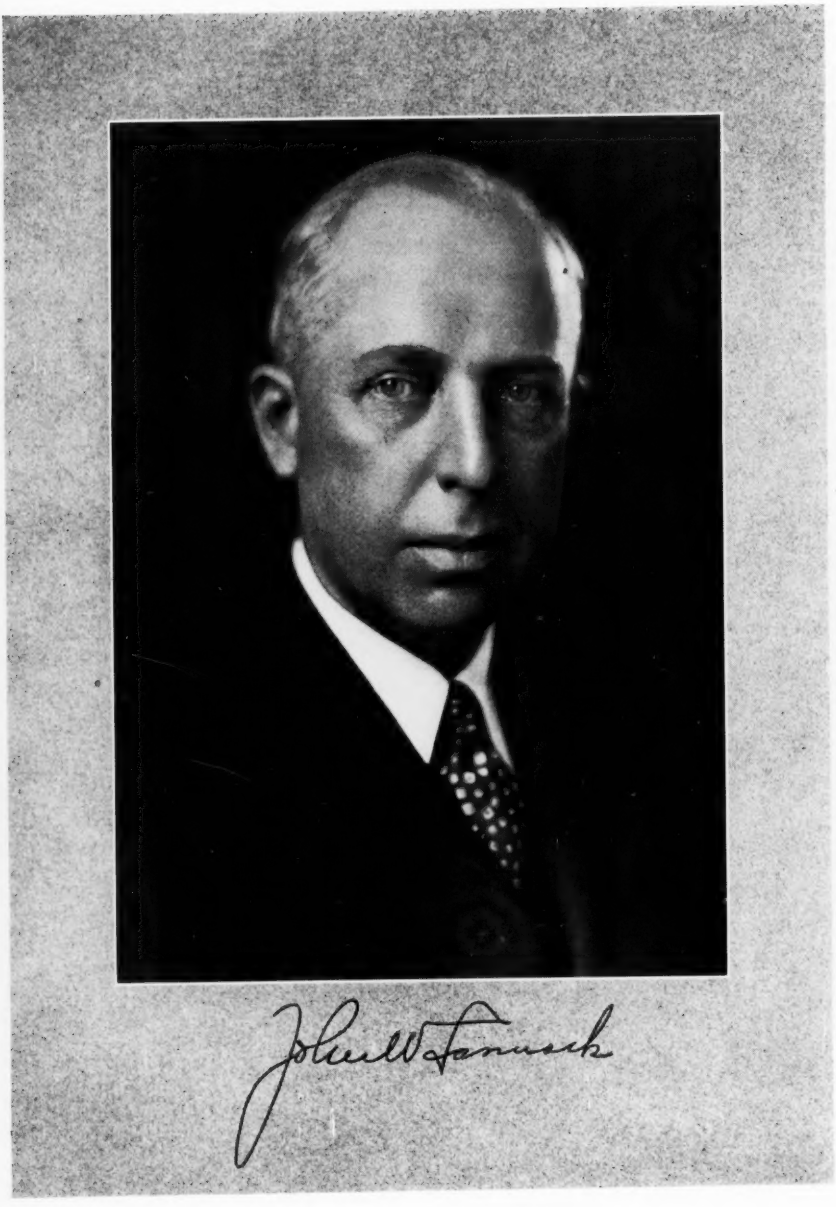
Besides serving as president of the American Medical Association, Dr. Phillips was president of the Medical Society of the County of New York in 1909 and president of the Medical Society of the State of New York in 1912. For twenty years he was Professor of Otology at the New York Post Graduate Medical School and Hospital; he was a Surgeon Director of the Manhattan Eye, Ear and Throat Hospital and for many years headed its ear department. He was a fellow of the American College of Surgeons, the American Otological Society, American Laryngological, Rhinological and Otological Society, and the American Academy of Ophthalmology and Otolaryngology.

In 1914 Dr. Phillips became an officer of the New York League for the Hard of Hearing, serving as a vice-president, as president and as a director for the last twenty years of his life. Through his connection with this pioneer organization, he became the founder, in 1919, of the American Federation of Organizations for the Hard of Hearing, serving as its first president. Under his leadership this federation grew from one member-organization in 1919 to nearly one hundred fifty in all parts of the United States and Canada at the present date. Prominent physicians, physicists, educators and social workers, both hard of hearing and normally hearing, were attracted to it and its nation-wide program for conservation of hearing, especially in childhood, when, as Dr. Phillips pointed out, impairments of hearing, frequently developing later into an economically crippling handicap, actually begin in the majority of cases. This school hearing program has been approved by the great medical societies and has been widely adopted in American municipal educational systems and is now being introduced into the New York City schools. During the last two years, Dr. Phillips had served as chairman of the Committee on the Deaf and Hard of Hearing, a subcommittee of the

Committee of Public Relations and of Public Health of the Medical Society of the State of New York, its objectives being to clear up the confusion regarding the deaf child, with its need for special education, and the hard of hearing child, who should be discovered, medically cared for and retained in the regular public schools with instruction in lip reading and appropriate vocational guidance.

During his service as a trustee of the American Medical Association Dr. Phillips was instrumental in the initiation of *Hygeia,* the health magazine, while his presidency, in 1926, was notable for his advocation of public health measures and the importance of the annual health examination. As president of the New York State Medical Society he urged the medical profession to enter into closer relations with the public for the furthering of health education. Dr. Phillips was the author of a textbook, "Diseases of the Ear, Nose and Throat," which is in use in eighty per cent of American medical schools, and of many contributions to medical and other publications on ear diseases, general health topics and social work for the hard of hearing. He was also co-author of "Your Hearing: How to Preserve and Aid It," a handbook for the general public.

Dr. Phillips was the son of Samuel and Mary Merrill Phillips of Hammond, St. Lawrence County, N. Y. He was a graduate of the Potsdam (New York) Normal School and of the Medical School of New York University in 1882. He married Miss Lucia M. Taggart of New York City, who survives him, with two daughters, Mrs. Craig F. Cullinan of Houston, Texas, and Mrs. Paul E. Johnson of Forest Hills, N. Y.



JOHN WALTER CARMACK.

1885—1934.

John W. Carmack, of Indianapolis, died suddenly on December 5th as the result of an aeroplane accident which occurred as he was returning from a medical meeting at Detroit.

Doctor Carmack was Professor of Otolaryngology and the head of the Department in the University of Indiana School of Medicine.

He was born and reared in Dana, Ind., where a number of relatives still live. He had been a resident of Indianapolis since he completed his work in the Indiana University School of Medicine, where he was graduated in 1907 and served his interneship in the City Hospital. He began his career as a general practitioner and became a specialist in ear, nose and throat diseases a short time before the World War. He served as First Lieutenant at Camp Taylor during the war.

Doctor Carmack was Secretary of the Section on Laryngology, Otology and Rhinology of the American Medical Association. He was a member of the Indianapolis Medical Society, the Indiana State Medical Association, the American Academy of Ophthalmology and Otolaryngology, and the American Laryngological Association. He was a Fellow of the American Medical Association and the American College of Surgeons and a member of the American Laryngological, Rhinological and Otological Society and the American Board of Otolaryngology.

Doctor Carmack was a member of the official board of the Boy Scouts in Indianapolis and a member of the executive committee. He was chairman of the health and safety committee and was instrumental in obtaining full-time medical attention at the Scout reservation during the summer camps.

He is survived by his widow, Mrs. Bertha J. Carmack, and a son, John W. Carmack, Jr., nine years old.

Abstracts of Current Articles.

NOSE

The Action of Cilia and the Effect of Drugs on Their Activity.

Negus, V. E. (London), J. of Laryng. and Otol., 49:571 (Sept.), 1934.

Stimulated by the recent interest in ciliary activity in the nose, the author was moved to study the effects of certain drugs, and particularly the pH of media upon their activity. In a brief resumé of the early observations upon cilia, he quotes Sharpey (1835): "Acid, alkaline and saline solutions when concentrated arrest the motion instantaneously . . . ." He stresses the effectiveness of the activity and points out that gravity, and hence the upright position of man, have little effect upon the cleansing of the respiratory tract.

He calls attention to the apparent discrepancy between the published figures of a pH of 6.4 in pus from suppurating sinuses, at which concentration cilia are said to be paralyzed, and the long series published by Proetz of active cilia in such cases.

Negus made many observations with solutions of sodium chloride, sodium acetate and Tyrode's solution with a pH of 9.0 down to 4.0. He found that when the pH was lowered to 6.4 or less, ciliary action did not continue, as contrasted with vigorous action after twenty-four hours at 8.5.

Nasal Flora and Reaction of the Nasal Mucus.

Tweedie, A. R. (Nottingham), J. of Laryng. and Otol., 49:586 (Sept.), 1934.

The author reports researches with a colorimeter for determining the pH values of secretions ranging between 6 and 8, and reports observations upon a series of two hundred cases. His most important inference is that until the reaction of the mucus becomes disturbed, pathogenic bacteria cannot obtain a footing.

The method consists of swabbing the anterior end of the middle turbinate and noting color effects of brom-thymol blue and cresol-purple on the mucus. Comparison is made with a standard color index.

Nasal Ciliated Epithelium, With Special Reference to Infection and Treatment.

Proetz, A. W. (St. Louis), J. of Laryng. and Otol., 49:557 (Sept.), 1934.

This very interesting and comprehensive paper represents the outcome of a series of investigations on the study of the cilia of nasal and sinus mucous membranes in animals and in humans. Ciliary activity was studied under both normal and pathologic conditions. Especial importance is placed upon the practical deductions which

may be drawn from the effects of drugs and physical changes. Another very practical observation was the effect of sinus infection upon ciliary activity.

Attention is called to the protective blanket of mucus which completely covers the surface of the membranes throughout the nose and sinuses and its remarkable protective powers. Changes in the viscosity and mucin content of this mucus layer are considered under normal and pathologic conditions. This layer is under constant motion as the result of ciliary activity and it has been estimated that it takes about thirty minutes for mucus to be carried from the farthest points in the nasal chambers to the nasopharynx.

These investigations have disclosed two important points, namely, that normally functioning ciliated epithelium is regenerated in the sinuses following the complete removal by radical operation, and that vigorous and functioning cilia may be found in the midst of hyperplasia and pus in chronic sinusitis.

In studying the local effects of drying the mucous membrane, it was found that there is a slowing of ciliary activity as the result of increased viscosity of the mucus, thus permitting the possible penetration of the bacteria.

In estimating the effects of heat and cold, Proetz found that the optimum temperature for ciliary activity is between 28 and 33 degrees C. When the temperature is lowered, ciliary activity ceases at about 7 to 12 degrees C., and when the temperature is elevated, ciliary activity ceases at about 43° C.

In the study of the effects of drugs, 130 observations were made upon intact and excised specimens from the rabbit and upon excised specimens from the human sinuses.

Ciliary activity continues for long periods in .9 per cent sodium chloride at temperatures between 25 and 30 degrees C. At a concentration of 4.5 to 5 per cent all activity ceases but may be restored after removing this solution and replacing with normal saline. When the concentration is lowered to .2 to .3 per cent concentration activity ceases but cannot be restored after replacing it with normal saline.

The application of liquid petrolatum does not interfere with ciliary activity because it remains on the surface of the mucus layer and does not come into actual contact with the cilia.

Extirpated membranes show ciliary activity for fifteen minutes or more when treated with 3 per cent ephedrin solutions. The living mucosa in the rabbit seems unaffected by this solution. No appreciable difference was found with the application of 2 per cent, one-half per cent and with ephedrin in oil.

As a rule, ciliary activity ceases immediately upon the application of 10 per cent cocain hydrochloride and may be resuscitated only in certain areas with difficulty. In extirpated specimens, 5 per cent cocain stopped ciliary activity within one to three minutes. Attempts to revive failed. Two and one-half per cent cocain stopped ciliary activity only after continuous application for one hour.

A 1/1000 solution of epinephrin caused immediate cessation of ciliary activity which could not be resuscitated. With a 1/5000 solution activity persisted for about twenty-five minutes. Upon application of a 1/10,000 solution for five minutes, activity persisted for two hours or more.

The effects of other agents such as camphor, thymol, menthol, argyrol, mercurochrome and merthiolate solutions were also reported, but attention was called to the fact that these experiments deal only with the rate and persistence of the ciliary beat, and that any generalization regarding relative therapeutic values, drawn from them alone, may be misleading.

On the basis of these experiments, Proetz makes the practical deduction that it seems quite improbable that the bacteria in infections penetrate the normal mucus layer. He believes that it is more probable that disabling occurs first through drying or some other of the agencies mentioned and that infection follows. The problem of the possible part played by anatomic deformities or abnormalities in relation to the infection and ciliary activity is considered.

It is suggested that new infection probably occurs through ciliated areas which are suddenly incapacitated rather than through thick, hyperplastic areas which are relatively impervious, and therefore it may be necessary to correct particularly those conditions which create transient aberrations of ventilation. It is generally known that the polypoid nose is often quite resistant to infection.

Proetz concludes his paper with the discussion of the problem of the treatment of the nose to promote the flow of normal mucus when it is lacking. He is convinced that an effective means of quickly restoring normal mucus to the surface will go far in the prevention and abortion of colds. HANSEL.

Surgical Treatment of Serious Epistaxis (Le traitement chirurgical des epistaxis rebelles).

Dufourmentel, L. (Paris), O. R. L. Internat., 18:492 (July), 1934.

The author resects enough of the cartilaginous septum underlying the bleeding area to permit cicatricial union of the latter with the healthy mucosa of the opposite side, and claims uniformly favorable results. FENTON.

The Olfaction at Various Ages (L'olfatto nelle diverse età).

Mesolella, V. (Naples), Arch. Ital. di Otol., Rino. and Laryng., 46:43 (January), 1934.

The author reports the results of his histologic investigation to determine if the olfactory sense is decreased or lost with the increase of age, due to the atrophy of the mucosa, the bulb, or the nerves, as maintained by Vaschide, Douglas, Prevost and Bilancioni.

The research was conducted by examining the maculæ luteæ, the olfactory bulb and the hippocampus of macrosmatic animals of various ages, and by examining the maculæ luteæ and the bulb of five individuals of advanced age. Later, with the aid of the olfactometer, he carefully measured the olfactory sense of sixty-eight persons of various ages. From experimental results he draws the following conclusions:

1. That in macrosmatic animals there is no structural change in the maculæ luteæ, the bulb or the hippocampus.

2. That in individuals of advanced age the olfactory bulb shows some rarefaction and atrophy, probably due to the destruction of peripheral neurones caused by catarrhal inflammation.

3. That in some aged individuals there is a slight decrease in the sense of smell, which is produced by chronic catarrhal inflammations, by stenosis of the nasal fossæ and by toxic substances, such as tobacco in the form of snuff or smoke.

4. That in children the olfaction limit is a little lower.

This article contains ten histologic illustrations and the tabulated results of the olfactometer readings. SCIARRETTA.

The Reactivating Action of Hypophyseal Extracts on the Changes of the Mucosa and Bones of the Nose Caused by Castration (L'azione reintegrativa degli estratti anteipofisari ed ovarici sulle alterazioni sperimentali della mucosa ed ossa nasali da castrazione).

Pighini, G., and Porta, C. F. (Parma), Valsalva, 10:140-143 (Feb.), 1934.

The author discusses the literature of castrated subjects in respect to changes occurring in the nasal and skeletal structures. He performed experiments on castrated rabbits and draws the following conclusions:

1. The postmortem examination of the castrated young animal shows macroscopically: Nasal bones are softer than normally and the sutures are not completely united. The anterior turbinate is enlarged and there are dystrophic changes of the cranial bones. Microscopically (ten microphotographs): The turbinates show the reduction of the bony framework proved by the bone absorption and its replacement by young connective tissue. The mucosa is flattened and fatty degeneration is evident. The glands are poorly developed.

2. The above described degenerations disappear almost completely with the administration of follicular liquor or anterior pituitary extracts.

3. The pathologic changes found in these animals improve considerably after the injection of the ovarian follicular liquor, and the histologic structures approximate the normal if the anterior pituitary substance is administered.

4. The above facts prove the interrelation of the nose and the gonads through the internal secretion of these glands.

SCIARRETTA.

Retrobulbar Neuritis, Sinusitis, Multiple Sclerosis (Névrite rétrobulbaire-sinusite-sclérose en plaques).

Weill, G., and Nordman, J. (Strasbourg), Rev. d'Oto-Neuro-Oph., 12:497 (July-Aug.), 1934.

The authors suggest that cases of optic neuritis which have been operated upon for posterior sinus disease should be followed up for several years to determine whether latent multiple sclerosis did not develop later. They suggest that the same micro-organism, entering by the bucconasal route, may be responsible for all three conditions; also that extensive drainage procedures in the nose are less likely to have late complications than mere puncture or washing out of these remote cavities. FENTON.

Mycotic Infections of the Maxillary Sinuses.

Stevenson, Holland N. (New Rochelle), Arch. of Otolaryng., 20:340 (Sept.), 1934.

The author has been searching for fungus infections in cases of sinus disease and in this article reports seven cases. The fungus forms discovered resemble those of mucor-histoides. Six cases were classed as chronic sinusitis. Four cases had recurrences while under observation. In five cases the fungus infection was bilateral. Observations indicated that fungi were present as active agents rather than saprophytes. TOBEY.

Acute Frontal Sinusitis with Enterococcus Exudate (Sinusite frontale acuta con essudato mucoso da enterococco).

Bronzini, A. (Pisa), Boll. delle Malatt. Orecch., Gola, Naso, etc., 52:27 (Jan.), 1934.

Reports a case of acute right frontal sinusitis caused by the enterococcus, which was cured by surgical procedure and autovaccinotherapy.

This is the first case of enterococcus sinusitis reported, as far as Bronzini knows. SCIARRETTA.

Chronic Maxillary and Ethmoidal Sinusitis in Children of Seven to Twelve Years (La sinusite maxillaire chronic avec sinusite ethmoidale chez les enfants de 7 à 12 ans).

Thorkildsen, V. (Oslo), Acta Otolar., 20:3-4:516 (1934).

Study of 204 children in Prof. Leegaard's clinics showed 128 with nasal stenoses and various symptoms of rhinitis; among these, sixteen had chronic maxillary and ethmoid involvement. Symptoms, mainly unilateral, included besides discharge, slight odor, headache and occasional low fever. Puncture usually confirmed radiologic diagnosis, but lavage oftener than every fourth day was found irritating. Blood sedimentation tests and determination of the bacterial flora seem to indicate that the sinusitis is not a true bacterial infection but rather that the retained mucus secretions should be considered as a propitious culture medium for germs of low pathogenicity, which latter are nevertheless capable of continuing an inflammatory condition of the mucosa. Conservative lavage, with boric or saline, vaccines and general hygiene are advised; adenoids must come out. Radical operation may become necessary but very rarely. FENTON.

PHARYNX

Cyst of the Epiglottis. Case Report (Considerazioni su di un caso di cisti dell' epiglottide).

Lopez, A. (Pisa), Boll. delle Malatt., Orecch., Gola, Naso, etc., 52:71 (Feb.), 1934.

Various theories are reviewed of laryngeal cysts genesis. The author reports a patient, aged 67, having a cyst, the size of a large walnut, located on the lingual surface of the epiglottis, which was grasped by a snare and excised by scissors. The histologic examination disclosed that the lining surface consisted of two to four layers of cuboidal or cylindrical epithelium, which in spots was covered with cilia. The origin of the cyst was due to obliteration of a crypt of the mucous membrane and not to the occlusion of a secretory gland duct. SCIARRETTA.

LARYNX

Malignant Disease of the Larynx: Rare Types, Premalignant Conditions, and Conditions Simulating Malignancy.

MacKenty, John Edmund (New York), Arch. of Otolaryng., 20:297 (Sept.), 1934. Edited by E. Ross Faulkner, M. D.

Excellent illustrations with descriptions of cancer, syphilis, tuberculosis and the rarer diseases of the larynx are given. Cancer in the young and aged is discussed and cases are reported of sarcoma, prolapse of Morgagni's ventricle (later diagnosed carcinoma), acromegaly, scleroma, blastomycosis, pemphigus and rare combinations. TOBEY.

The Protracted-Fractional X-ray Method (Coutard) in the Treatment of Cancer of the Larynx.

Webster, J. H. Douglas (London), J. of Laryng. and Otol., 49:429 (July), 1934.

The article from the radiologic department of Middlesex Hospital, London, discusses the progress and the most recent advancements in the technic of x-ray therapy for advanced cases of cancer of the larynx. The different stages in the progress of technic and the disadvantages of the methods of Coutard, Finzi and Holfelder are given. The writer believes that the only method which can show convincingly good results, after long periods of years, is that of Coutard, and its value has also recently been confirmed by the results of a number of other radiologists. He gives five essentials in this method of treatment. Tables are given showing the results with x-ray therapy, and with the method of Coutard they show a percentage of 32 per cent (three years) of seventy-seven cases.

The author believes that we are now at the beginning of a really great advance in x-ray therapy. Critical consideration of the evidence that exists shows conclusively that it gives great promise for the future usefulness of the x-ray branch of radiation therapy, at least in squamous celled carcinoma of the larynx, in pharyngeal, tonsillar and probably some other squamous celled carcinoma and possibly for other sites and types of cancer. GOLDSMITH.

EAR

Discussion on the Treatment of Chronic Catarrhal Otitis Media (Excluding Otosclerosis) and of Deafness and Dry Suppurative Otitis Media.

Proc. of the Royal Soc. of Med., Vol. XXVII, No. 8, page 1069.

Mr. W. M. Mollison, in opening the discussion, referred to the gloomy prognosis in chronic catarrhal otitis media and pointed out that the treatment, to be effective, must be largely concerned with the beginning of the disease, though one may help the chronic cases that suddenly become worse owing to reinfections. Chronic catarrhal otitis media and dry suppurative otitis media are the results of previous subacute or acute inflammation in the middle ear tract. In the first, perforation of the membrane has occurred, and there has been suppuration, persisting for a shorter or longer time. Just as acute suppurative otitis media leads to perforation of the membrane and later to dry perforations, so acute catarrhal otitis media leads to exudative otitis media, acute or subacute, and, if unrelieved, results in chronic catarrhal otitis media. If the catarrh is a mild one and in a child, attention is not drawn to it as there may be no pain, or pain so transient that it is disregarded, and certainly the deafness is not noticed. Once established in children, a catarrhal otitis media leads to increasing deafness, compensated for in the young, but be-

coming worse with age. The discovery of deaf children now being made with the use of the audiometer in the examination of school children will lead to treatment and doubtless to a diminution of chronic catarrhal otitis media. From the standpoint of deafness he believed it was better to have a suppurating ear than a catarrhal one.

The diagnosis of this disease is sometimes easy, sometimes very difficult. There are cases in which the membrane is often substantially normal, though sometimes lacking the normal sheen and typical normal appearance. The typical sound of fluid is easily heard, but he suggested that a too easily heard sound by inflation with the catheter indicates exudation.

In the treatment the fluid must be removed. If the Weber Liel tube, which is passed through the catheter into the tympanum and suction applied, does not succeed in this, then incision of the membrane should be performed and the patient made to perform Valsalva's self inflation. It is this "hidden fluid" group that escapes diagnosis, and therefore treatment passes on to chronic catarrhal otitis media. Several illustrative cases are given to show that in children one need not despair of recovery of hearing, even after long periods, and that sources of infection must be removed both from the nasopharynx and sinuses. One case given suggests that exudative otitis media may pass to the antrum and mastoid cells as in suppurative cases. Inflation of the tube and passage of a bougie as well as other treatment of the tube and membrane are reviewed. Unless we can treat effectively the early stages of catarrhal otitis by removing infective foci in the nasopharynx and accessory sinuses, in addition to removing the exudate, we shall not have progressed further than did our predecessors of sixty years ago. By prompt treatment of suppuration in the middle ear we may hope to eliminate "dry suppurative otitis media."

Mr. C. P. Wilson said that in his opinion cases of catarrhal deafness with atrophic changes of the membrani tympani and widely open fibrotic eustachian tubes have also the condition of otosclerosis. In these cases any active local treatment is more likely than not to increase the rate of progress of the disease. The only hope for these patients is to see that the infective process in the nasopharynx damages the ear no further, and improve the compensation. He suggested that the anatomy of the eustachian tube is a factor of more importance than we usually consider. The normal tube is a long, narrow tube, but there is a small percentage of people who have a short, relatively wide tube. The latter class is more vulnerable to the spread of infection from the nasopharynx, but the tube rarely becomes obstructed unless the infection is a virulent one. The ciliary action of

the eustachian tube is sufficient to help the drainage of the middle ear. People with short, wide tubes have frequent attacks of mild subacute catarrh with very little, if any, pain associated with the attacks because there is no distension of the middle ear. Patients with long, narrow eustachian tubes have fewer attacks of infection from the nasopharynx, but the attacks that they do get are associated with eustachian obstruction, exudate in the middle ear, and, possibly suppuration. It is in these cases that local treatment is of value. Catheterization should be undertaken only when there is definite eustachian obstruction and only continued if it results in definite improvement of hearing.

Dr. Albert A. Gray reviewed the pathologic changes in the conditions under discussion. Adhesions which may occur after an acute or subacute middle ear inflammation which has not gone to the stage of suppuration, may be found in various regions of the middle ear, but are perhaps most common about the head of the malleus and body of the incus. Adhesions which may be found after the drying up of a chronic middle ear suppuration not infrequently bind down the remains of the tympanic membrane to the promontory. There is another condition in which newly formed fibrous tissues may be found in the middle ear, without any preceding inflammation or exudation, and consists of a very slow increase of the connective tissue in the mucoperiosteum of the middle ear. It is not inflammatory in character but is a degenerative process and from his investigations he has never found it except in association with otosclerosis. Inflation in these cases can do no good and very probably will do harm.

Mr. Thacker Neville believed that in the case of chronic catarrhal deafness anything which dilated the blood vessels of the inner ear seemed to help. He used diathermy, by which he applied heat to the ears for twenty minutes, as a routine in all cases of catarrhal deafness.

Dr. A. R. Friel said that any treatment which is effective for rhinitis in children is a preventive or curative agent for the ear condition and referred to the treatment of Dr. Gautier of Paris, called diastolization. Acute rhinitis may readily become chronic owing to retention of infected mucopurulent secretion in the posterior part of the floor of the nose. Diastolization consists in the introduction of soft rubber bougies along the floor of the nose into the space between the inferior turbinal and septum. The mild stimulus causes, by reflex action, the opening of the passage and the secretion is expelled.

Mr. T. B. Layton suggested that changes in and around the mastoid antrum played some part in the cause of deafness. If the middle ear cleft was a part of the organ of hearing then the furthest end of

the diverticulum must have something to do with this function. A person suffering from otitis media was not usually deaf, but if he were so it was certain that the inflammation had extended into and involved the tissues of the mastoid region.

Mr. Eric Watson-Williams said there was a definite group in which the trouble seemed to be a degenerative process affecting the mucous membrane of nose, pharynx and ear (he was not referring to the mucosal degeneration of otosclerosis). These patients appeared to be suffering from iodin deficiency, and he had obtained a certain amount of success by treating them with thyroid gland substance.

Mr. Harold Kisch gave a test which he had used in deciding whether or not to perform submucous resection or other operation for nasal obstruction in cases of chronic deafness. He tested the hearing of a patient before carrying out any manipulation, and then sprayed the nose with a solution of cocain and adrenalin. In a certain number of cases the hearing could be demonstrated to have improved, and in these he had found that operation for the nasal obstruction was helpful.

Mr. J. F. O'Malley said that what seemed necessary in order to improve treatment was to gain a better understanding of the physiology of the air movement influences in the middle ear cavity, which was essentially an air cavity, with variations of pressure from time to time. If the maintenance of the air contents in these cavities was efficient and capable of varying, hearing seemed to be satisfactory. Conditions which altered these seemed to be conditions which deteriorated the functions of the ear. GOLDSMITH.

Experiments with Venosinography in Sinus Thrombosis.

Frenckner, P. (Stockholm), Acta Otolar., 20:3-4:477 (1934).

Expressing the opinion of the Holmgren clinic that the Tobey-Ayer test is unreliable, Frenckner has trephined dead monkeys and human beings, and live monkeys, over the middle of the longitudinal sinus while on the x-ray table, injecting 10 cc. of thorotrast by a special cannula at the rate of 2 to 3 cc. per second and taking several films during the last third of the injection, after which the cannula is rapidly removed. Delay of two to three seconds means disappearance of the opaque material from the suspected area. With an occipital plate both sinuses are shown at once and areas of occlusion readily demonstrated. This method, contrary to that recently reported by O. J. Dixon, does not enter a sinus which may contain infected clot but places the radiopaque solution in a clean vein far above. Human experiment will demonstrate the value of this procedure, especially during incomplete thrombosis. FENTON.

Experiments on Eustachian Tube Function (Recherches sur les fonctions de la trompe d'Eustache).

Holmgren, G. (Stockholm), Acta Otolar., 20:3-4:381 (1934).

Prof. Holmgren has determined that ligation of the dog's eustachian tube brings down air pressure in the tympanic cavity continuously; this experiment lasted one and one-half hours. After several days either retraction of the membrana tympani or an accumulation of mucoid secretion within the drum cavity is noted. This exudate is wholly or in large part due to retention of glandular secretion from the tympanic mucosa. Xeroform powder or grains of starch introduced into the tympanum were carried into the nasopharynx by ciliated epithelium in a very brief time, as shown by inspection of the tube mouth. FENTON.

MISCELLANEOUS

Ménière's Disease: Symptoms, Objective Findings and Treatment in Forty-two Cases.

Dandy, Walter E. (Baltimore), Arch. of Otolaryng., 20:1 (July), 1934.

The signs and symptoms in forty-two cases of Ménière's disease are analyzed. The disease occurred twice as frequently in males and twice as frequently on the left side. Vertigo, deafness and tinnitus were the first symptom in about equal percentages. The diagnosis, differential diagnosis and character of Ménière's disease are discussed. An operation of severing the vestibular portion of the eighth nerve while preserving the cochlear function is advocated and described in detail. TOBEY.

Recent Advances in the Treatment of Carcinoma of the Esophagus From a Surgical Aspect.

Turner, Grey (Newcastle-on-Tyne), J. of Laryng. and Otol., 49:297 (May), 1934.

For twenty-five years the author has carried on research work on surgery of the esophagus. He reviews in this article the treatment of this disease and gives in detail a case where he performed the radical removal of the esophagus by the method which he has designated as the "pull through" method. Later a new esophagus was constructed by making a tube from the skin over the front of the chest, the lower end of which was completed by an isolated loop of jejunum with which the gap between the skin tube and the stomach was bridged. By this means the patient was able to take all food by the mouth.

In this operation there are details of great importance which must be observed if it is to be successful.

The author believes that radical surgery seems to hold out some promise in these cases. In the past there has been little attempt to deal with these cases radically at an early stage. If we could overcome

the technical difficulties and resort to radical surgery at an earlier stage they might be more often treated sucessfully. It is better not to attempt the repair of the esophagus in situ, but after recovery from the excision to supply the defect by making a new esophagus by the anterior route. For growths of the upper end he suggests an excision with formation of a cervical fistula and completion by a new esophagus. For the lower end the writer would adopt the plan as described in the case reported, but for the middle portion he is inclined to give consideration to the posterior mediastinal or the transthoracic route. Information as to the exact living anatomy of these growths is required from the radiologists and those who make esophagoscopic examinations. GOLDSMITH.

Vasomotor Variations of the Skin Due to Thermic Stimulation of the Upper Respiratory Tract (Sulle variazioni vasomotorie della cute da stimoli termici delle prime vie aeree).

Muzio, O. (Genova), Boll. delle Malatt. Orecch., Gola., Naso., 52:12 (Jan.), 1934.

The author reviews briefly the nerve supply of the nose and discusses reports published on the subject by various researchers. While these other workers have produced thermic changes of the skin and later registered the thermal variations of the expired air, Muzio instead carried his experimental studies of the vasomotor reactions on the individual (age 20 to 35) by registering the thermic changes on the skin of the arm after the inhalation of warm (50° C.) or cold (0° C.) air through the nose alone or the mouth. His conclusions are the following:

1. Thermic excitation of the upper respiratory routes demonstrates always a vasomotor reaction of the skin.

2. The vasomotor reactions produced by the stimulations of the nasal mucosa are more prompt and more intense than those produced by the stimulations of the mouth and laryngeal mucosa.

3. In general whenever a current of warm air passes through the nose or the mouth a cutaneous vasodilatation is registered, and vice versa, with the cold air, vasoconstriction results.

4. These reactions prove that the upper portion of the respiratory tract possesses a thermoregulator system which acts through the sympathetic and parasympathetic nervous systems.

SCIARRETTA.

Books Received.

Recent Advances in Allergy (Asthma, Hay Fever, Eczema, Migraine, etc.).

George W. Bray, M. B., Ch. M. (Sydney), M. R. C. P. (London), Physician in Charge of Children's Department, Prince of Wales Hospital; Assistant Physician, Princess Elizabeth of York Hospital for Children; Clinical Assistant, Asthma Clinic, Guy's Hospital; Late Asthma Research Scholar, the Hospital for Sick Children, Great Ormond Street, with foreword by Arthur F. Hurst, M. A., M. D. (Oxon), F. R. C. P. Senior Physician, Guy's Hospital; Chairman, Medical Advisory Committee, Asthma Research Council of Great Britain. Second Edition. Cloth. Octavo of 503 pages with 106 illustrations including four colored plates. Philadelphia: P. Blakiston's Son & Co., Inc.: 1934. Price $5.00.

Five hundred pages, packed with terse and accessible information, well illustrated. In this day, when every mail brings a new suggestion for the management of some phase of allergy, it behooves the laryngologist to ground himself in the currently accepted principles.

This book is one of the best manuals on any subject which have come to our notice.

Tuberculosis of the Lymphatic System.

Richard H. Miller, M. D., F. A. C. S., Assistant Professor of Surgery, Harvard Medical School; Associate Surgeon, Massachusetts General Hospital. Cloth. Octavo of 248 pages with 29 illustrations. New York: The Macmillan Company: 1934. Price $4.00.

This short and concise monograph approaches the subject from the clinical standpoint and should be especially valuable to those whose work touches in some way on the disease without concerning itself wholly with it. This for the reason that the material is accessible and complete and is largely stripped of controversial matter.

The book is recommended to laryngologists for two reasons: First, because it concerns them clinically; second, because it emphasizes once more the need for study of the lymphatics of the head and neck from the standpoints of invasiveness and penetrability, and as avenues of elimination and loca of retention in the less violent nose and throat infections.

Principles and Practice of Otology.

F. W. Watkyn-Thomas, F. R. C. S., B. Ch. Camb., Surgeon, Central London Throat, Nose and Ear Hospital, and A. Lowndes Yates, M. C., M. C. Lond., F. R. C. S., Edin., Honorary Assistant Surgeon, Ear and Throat Department, Prince of Wales Hospital; Honorary Assistant Surgeon, Central London Throat, Nose and Ear Hospital. Cloth. 8vo of 555 pages with 199 illustrations (five colored). Baltimore: William Wood & Company: 1933. Price $8.25.

Osteomyelitis. Its Pathogenesis, Symptomatology and Treatment.

Abraham O. Wilensky, A. B., M. D., F. A. C. S. Fellow of American Association for Thoracic Surgery; Attending Surgeon to the Bronx Hospital and Dispensary; Associate Attending Surgeon to the Mount Sinai Hospital. Cloth. Octavo of 454 pages with 104 illustrations. New York: The Macmillan Company: 1934. Price $9.00.

This work tells the story of osteomyelitis from what must have been about the beginning of bones to the present day. Chapters dealing with the anatomy of bone, and the mechanism of its invasion by pathogenic organisms, should be extremely instructive to anyone so constantly involved in bone surgery as is the otolaryngologist.

Of special interest to him are the seventy pages devoted to osteomyelitis of the skull. In his discussion of the types of frontal sinus operations the author very properly warns against the non-obliterating frontal wall type of operation, although rhinologists will scarcely agree with him that this is the type of operation most generally employed.

Your Hearing. How to Preserve and Aid It.

Wendell C. Phillips, M. D., Consulting Surgeon, Manhattan Eye, Ear and Throat Hospital, New York City; Founder, American Federation of Organizations for the Hard of Hearing; and Hugh Grant Rowell, M. D., Assistant Professor of Health Education and Physician to the Horace Mann Schools, Teachers College, Columbia University. Cloth. Sexto-decimo of 232 pages with 11 illustrations. New York: D. Appleton and Company: 1932. Price $2.00.

Index of Authors.

Index of Titles.